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# Acute Alitasic Cholecystitis

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## Abstract

Acute acalculous cholecystitis (AAC) is the inflammatory disease of the gallbladder in the absence of gallstones. Typically affects critically ill patients. Diagnosis is not straightforward as Murphy's sign is difficult to detect in critically ill and many imaging findings are numb or nonspecific. Acalculous cholecystitis is a life-threatening disorder that has a high risk of perforation and necrosis compared to the more typical calculous disease. Management involves a percutaneous cholecystostomy, a surgical cholecystectomy, or, more recently, a metal stent placed endoscopically through the gastrointestinal tract into the gallbladder. Acalculous cholecystitis is a serious illness that has high morbidity and mortality. The reported mortality of the condition varies from 30 to 50% depending on the age of the patient. Even those who survive have a long recovery that can take months.

**Keywords:** cholecystitis, gallbladder, alithiasic

## 1. Introduction

Acute alithiasic cholecystitis is defined as an inflammatory disease of the gallbladder in the absence of gallstones or obstruction of the cystic duct and it has a multifactorial pathogenesis [1].

It accounts for approximately 10% (range, 2% -15%) of all cases of acute cholecystitis. Acute alithiasic cholecystitis occurs in approximately 0.2% -0.4% of all critically ill patients. Duncan recognized it, for the first time, in 1844 when a fatal case of acute cholecystitis complicating an incarcerated hernia was reported [2]. Acute cholecystitis acalculous is associated with a morbidity more serious and a rate mortality more elevated compared to acute lithiasic cholecystitis [3].

The death rate depends primarily on the presentation already critical of the patient as the disease affects both medically and surgically compromised patients. Clinically, acute alithiasic cholecystitis is indistinguishable from acute lithiasic cholecystitis. Many patients with acute cholecystitis acalculous have the same symptoms of gallstone cholecystitis: pain in the quadrant abdominal upper right, fever, neutrophili leukocytosis, elevated liver enzymes (ALT, AST, Alkaline phosphatase) increased serum total bilirubin and fractional [4].

There are various risk factors that predispose to the formation of acute alithiasic cholecystitis which are listed in **Table 1**, as noted this pathology mainly

Risk factors commonly associated with acute alithiasic cholecystitis	Risk factors rarely associated with acute alithiasic cholecystitis
Severe trauma leading to hospitalization; some factors particularly useful for the diagnosis of acute alithiasic cholecystitis are blood transfusions (over 12 units) and cardiac arrhythmias.	Hypovolemia
Recent cardio-pulmonary surgery	Cholangiopancreatography Endoscopic retrograde
Shock of any kind	Longer of hospital stay
Severe burns	Immunodeficiency: acquired immunodeficiency syndrome, organ transplant
Bacterial or viral sepsis	Chronic disease: diabetes, hypertension, atherosclerotic disease, morbid obesity
Critical illness (any patient requiring mechanical ventilation in the ICU)	Vasculitis: Churg- Strauss, giant cell arteritis, Henoch-Shonlein purpura, polyarteritis nodosa, lupus.
Total parenteral nutrition	
Prolonged fasting	

Owen & Jain [5] table.

**Table 1.**  
*Descending order of risk factors associated with acute alithiasic cholecystitis.*

affects patients in serious clinical conditions: severe trauma, patients with shock of any type on mechanical ventilation, with sepsis, burns or in total parenteral nutrition [6].

2. Pathophysiology

The etiology of acute cholecystitis acalculous is multifactorial, but is mainly formed by biliary stasis or organ wall ischemia. Biliary stasis can be caused by fast-ing, post-surgical total parenteral nutrition that leads to an increase in bile viscosity which irritates the mucous membrane of the gallbladder. Gallbladder wall ischemia occurs due to decreased blood flow due to fever, dehydration, or heart failure, which leads to the pathogenesis of acute cholecystitis [7].

It arises acutely when the walls of the gallbladder become inflamed for the reasons mentioned above [8].

Prolonged ischemia of the gallbladder walls leads to gangrene and then perfora-tion. If the process occurs slowly, the formation of cholecystoduodenal (70%), cho-lecystocholic (10–20%), and the less common cholecystogastric fistula is possible. This will lead to sepsis and shock. These findings are referred to as acute cholecys-titis. Chronic acalculous cholecystitis usually presents more insidiously. Symptoms are more prolonged and may be less severe. Symptoms may also be more intermittent and vague, although patients can present with signs of acute biliary colic [9].

3. Epidemiology

Acalculous cholecystitis has an incidence rate of 0.12% in the entire population. Rates are increased in HIV and other immunosuppressed patients. These indi-viduals are more susceptible to certain opportunistic infections such as microspo-ridia, cytomegalovirus (CMV), and Cryptosporidium, which can seed and flourish in bile within the gallbladder [10].

Carriers of *G. lamblia*, *H. pylori*, and *S. typhi* are also associated with increased risks to develop cholecystitis.

It can occur in all breeds. Acute alithiasic cholecystitis has a slight male predominance (80% of case), unlike acute lithiasic cholecystitis, which has a female predominance and occurs at any age with a criticality threshold between the fourth and eighth decade of life [11].

#### **4. Prognosis**

Acalculous cholecystitis is a serious illness that has high morbidity and mortality. The reported mortality of the condition varies from 30 to 50% depending on the age of the patient. Even those who survive have a long recovery that can take months [12].

#### **5. Mortality/morbidity**

The rates of the mortality and morbidity associated with acute cholecystitis can be high; the disease is frequently seen in patients with sepsis or other serious conditions. The reported mortality range is 10% - 50% for acute cholecystitis acalculous compared to 1% for acute cholecystitis lithiasic [13].

#### **6. Complications**

Perforations or gangrene of the gallbladder and extrabiliary abscess formation in the acute alithiasic versus lithiasic gallbladder may occur [14].

#### **7. History and physical Exam**

Often these patients are very seriously admitted to intensive care in mechanical ventilation and cannot participate in an anamnestic interview and therefore communicate their symptoms. Physical examination may detect fever, tenderness on the upper abdominal quadrants of the right associated with laboratory abnormalities such as neutrophil leukocytosis and altered liver tests (high values for ALT, AST, alkaline phosphatase and direct bilirubin) [15].

#### **8. Diagnosis**

The diagnosis of acute cholecystitis acalculous is difficult because no clinical data (symptoms, examination goal, testing laboratory) establish it. Although no combination of clinical factors will lead to the diagnosis, there seems to be a consensus on the fact that a high clinical suspicion for acute cholecystitis acalculous is indicated in all critically ill patients for whom no etiology has been found. The final diagnosis of acute cholecystitis is mainly based on radiological and ultrasound findings [16–18].

#### **9. Radiology**

There is controversy about what is the best imaging modality and which to use in the diagnosis of cholecystitis acute acalculous. However, radiological criteria for the

Mode	Criteria	Diagnosis
Abdominal ultrasound	Major: 3.5- to 4-mm (or more) thick wall (if at least 5-cm distended longitudinally with no ascites or hypoalbuminemia) Pericholecystic fluid (halo)/subserosal edema Intramural gas Sloughed mucosal membrane	2 major or 1 major and 2 minor (most studies have favored the diagnostic triad—wall thickness, sludge, hydrops)
	Minors: Echogenic bile (sludge) Hydrops distension greater than 8-cm longitudinally or 5-cm transversely (with clear fluid)	
TC	Major: 3- to 4-mm wall thickness Pericholecystic fluid Subserosal edema Intramural gas Sloughed mucosa	2 major or 1 major and 2 minor
	Minors: Hyperdense bile (sludge) Subjective distension (hydrops)	

**Table 2.**  
*Imaging criteria.*

diagnosis of acute alithic cholecystitis have been developed for the use of ultrasound and computed tomography. MRI is not used because it is a lengthy procedure with no benefit compared to the other modalities [19]. CT offers few advantages over ultrasound abdominal, unless there are other intra-abdominal pathologies that cannot be studied with the ultrasound. Therefore, abdominal ultrasound was the first line for the diagnosis of acute alithiasic cholecystitis as it can be performed at the bedside and favors patients who are intrasportable [20]. The ultrasound criteria for diagnosing acute alithiasic cholecystitis are: the thickness of the gallbladder wall, dangerous cystic fluid, wall, edema intramural gas, desquamated mucosa, mud or hydrops. The thickness of the gallbladder wall (3.5–4 mm) has been considered a crucial component for the diagnosis of acute alithiasic cholecystitis. Therefore, abdominal ultrasound is a very useful tool for diagnosing acute alithiasic cholecystitis as many prospective studies have suggested and, also, it is easy to use, fast, portable and easily repeatable at the bedside [21–23].

CT is useful for diagnosing acute alithiasic cholecystitis and other abdominal diseases. it requires patient transport, which may not be feasible, and offers few advantages compared to abdominal ultrasound. However, with a normal ultrasound, CT can diagnose acute alithiasic cholecystitis and make a differential diagnosis (**Table 2**) [5, 24, 25].

10. Therapy

The two prevalent treatment options for acute alithiasic cholecystitis are cholecystostomy (gallbladder drainage) and/or cholecystectomy. Other methods such as ERCP using stents or tubes have been tried but unsuccessful. Cholecystectomy is generally considered the definitive therapy. Some authors propose cholecystostomy as the only treatment. Others claim that the cholecystostomy is just a bridge to the cholecystectomy more secure or just a treatment to see if the acute cholecystitis acalculous is resolved. Therefore, Boland et al. recommend the cholecystostomy prophylactic for all intensive care patients with abdominal sepsis who do not improve with medical therapy (high-dose antibiotic therapy) [26, 27].

The cholecystostomy is generally plausible, quick and safe; it can be performed transperitoneally or transhepatically under ultrasound or CT guidance by interventional surgeons or radiologists. Therefore, cholecystostomy can provide time to optimize the patient's condition for cholecystectomy surgery. There seems to be an unanimous tendency to favor the cholecystostomy before cholecystectomy, unless there is a strong evidence of an ischemic cholecystitis that the drainage alone does not alleviate.

Cholecystectomy is a definitive therapy when performed by open or laparoscopic surgery. Laparoscopic surgery has been favored in recent years because it can be both diagnostic and therapeutic, it is less invasive, and it has similar morbidity and mortality compared to open procedures [28–31]. However, it should be noted that it may need to be converted to an open cholecystectomy and this should not be considered a failure of the surgeon on the contrary, when faced with situations in which it is not possible to distinguish, due to the inflammatory state of the gallbladder, the various structures anatomical, conversion to “open surgery” is preferable [32–35].

## 11. Conclusions

When an acalculous acute cholecystitis is suspected, the cholecystostomy must be carried out immediately, because the patient can only improve with this technique. If the improvement occurs with the decompression and drainage through cholecystostomy, the tube can be removed after 3 weeks, and this is the only treatment needed [36–39]. If there is no improvement, urgent cholecystectomy should be strongly considered as it can save the patient's life and thus improve abdominal sepsis [40–42].

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