Acalculous cholecystitis presentation in a young patient

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Abstract

Acalculous cholecystitis accounts for only 5-10% of cholecystitis cases and is often associated with severe trauma, critical illness, or chronic disease. Our case describes an otherwise healthy 25-year-old female presenting with acute abdominal pain. After undergoing a magnetic resonance cholangiopancreatography and cholescintigraphy she was diagnosed with acute acalculous cholecystitis. Her symptoms resolved following laparoscopic cholecystectomy which highlights the importance of prompt diagnosis and treatment of acalculous cholecystitis even in the absence of trauma or critical illness.

Keywords: Acalculous, Cholecystitis, cholecystectomy

Introduction

Acalculous cholecystitis is described as cholecystitis due to dysfunction and hypokinesis of gallbladder emptying. Often cholecystitis is caused by mechanical blockage due to a gallstone, however, in acalculous cholecystitis this is not the case. Individuals presenting with acalculous cholecystitis often have concomitant severe illnesses involving major surgery or intensive care treatment at the time of symptom onset. [1] The following describes a unique case of acute acalculous cholecystitis (AAC) in a young healthy patient with no significant predisposing conditions, trauma, or surgery.

Case

A 25-year-old female with a previous medical history of the polycystic ovarian syndrome, anxiety, and headaches came to the emergency department with abdominal pain of 2 days duration. She described the pain as sharp and constant, located in the epigastric and right upper quadrant region with no fevers, chills, nausea, or emesis. The patient stated a history of allergies to gluten and lactose and reported to be on a vegetarian diet. The patient also complained of loose nonbloody stools for 1 month before the current presentation.

On arrival to the surgical services, the patient had a temperature of 97.8 F and blood pressure of 127/82. Laboratory studies were significant for a white blood cell count of 10.8 K/uL and normal liver function test including aspartate aminotransferase (AST) 16, alanine aminotransferase (ALT) 27, alkaline phosphatase 46, and total bilirubin of 0.5. Additionally, respiratory syncytial virus (RSV), COVID-19, and influenza A and B were negative. Physical exam was significant for epigastric and right upper quadrant tenderness on palpation with voluntary guarding and no rebound tenderness. Ultrasound showed mild gallbladder sludge with no gallstones. Mild gallbladder wall thickening was also visible on ultrasound with a small amount of pericholecystic fluid [Image 1]. These results were indeterminate for acute cholecystitis. Mild gallbladder thickening with pericholecystic fluid was also seen on computerized tomography (CT) scan [Images 2a and b].

The patient was then admitted to the general medical floor and placed on nothing by mouth for observation and scheduled to undergo a magnetic resonance cholangiopancreatography (MRCP) and cholescintigraphy [(hepatobiliary iminodiacetic acid (HIDA)] scan. MRCP the day after admission revealed diffuse gallbladder wall thickening and mild pericholecystic edema. No gallbladder reported to be on a vegetarian diet. The patient also complained of loose nonbloody stools for 1 month before the current presentation.

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Stones were identified. Subsequent HIDA scan showed nonvisualization of the gallbladder at 1 h 90 min, 2, and 3 h postexam indicating acute cholecystitis.

Following HIDA scan results, the patient continued to be severely symptomatic. She underwent a laparoscopic cholecystectomy and was then discharged home the following day without complications. The pathology report revealed a gallbladder with hemorrhagic serosa, without lesions, and a gallbladder wall varying in thickness from 0.3 cm to 0.5 cm [Images 3a and b].

The patient continued to improve at 2 weeks follow-up reporting markedly improved ambulation, good appetite, and resolution of gastrointestinal pain.

Discussion

Acute acalculous cholecystitis (AAC) accounts for only 5%–10% of cholecystitis cases and is often associated with severe trauma, critical illness, or chronic disease. AAC is more commonly seen in males following surgical procedures complicated by hypotension and blood loss. Chronic illness can predispose individuals to AAC such as diabetes, autoimmune disorders, and congestive heart failure. The condition can also develop secondary to systemic illness involving bacterial, viral, or fungal infections with a recent study noting an association between AAC in young women and Epstein-Barr virus (EBV) infection. However, unlike our case, these individuals had a significant increase in liver function enzymes at the time of presentation. Diagnosing AAC can be a unique challenge, especially among young healthy patients with no predisposing factors, as seen in our case. Physical examination and laboratory studies are unreliable in the diagnosis of AAC including many nonspecific signs and symptoms such as leukocytosis, jaundice, and fever. Confirmation is often deferred from radiological studies with ultrasound being the modality of choice. After initial equivocal ultrasound results, our patient underwent an HIDA scan as well as a CT scan aiding as further evidence of AAC.

Our case is distinctive for displaying AAC in a young healthy woman with no severe illness, trauma, and without evidence of acute infection. Few studies have examined AAC in healthy patients with no predisposing conditions. One study examined AAC in four young healthy patients ranging in ages 26–32 challenging prior notions of this condition. More recent reports have noted 77%–90% of patients with AAC without acute illness or trauma. These findings highlight the importance of high suspicion of cholecystitis even in the absence of evidence of critical illness or gallbladder stones. A retrospective review examining AAC in the outpatient setting in individuals with no current critical illness found right upper quadrant pain to be the most consistent complaint. They also found most of the participants did not display a fever on presentation. Likewise, the average length of admission to operation, postoperative stay, and complication rate were comparable to that of routine calculous acute cholecystitis. These observations are consistent with our case.

Theories examining the pathogenesis of AAC commonly involve bile stasis leading to changes in chemical composition, sepsis, and ischemia. This phenomenon has been thought to arise in critically ill patients who are unable to tolerate oral intake and therefore have no stimuli for the gallbladder to contract leading to biliary sludge. There is additional evidence the pathogenesis surrounding AAC involves vascular distribution. When microangiography was observed in different causes of cholecystitis, dilated arterioles and a dense capillary network were seen in gallstone-associated cholecystitis; however, the irregular filling was observed within AAC. These findings suggest ischemia as a major factor in AAC. Likewise vasoactive mediators play a role in AAC pathophysiology including those produced during inflammation and infection.

There is increasing evidence that previous conceptions of AAC as a disease of trauma and critical illness are incomplete. Recent
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Studies have reflected an increase in AAC in the outpatient setting. Our case reflects these findings as well as challenging previous beliefs that AAC is mainly a disease of the elderly and critically ill. Understanding the conditions surrounding AAC is important for the prompt diagnosis and treatment of cholecystitis in individuals without evidence of trauma, critical illness, or gallstone involvement.

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Conflicts of interest
There are no conflicts of interest.

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