ARCHIVES OF HELLENIC MEDICINE: ISSN 11-05-3992

CASE REPORT
ΕΝΔΙΑΦΕΡΟΥΣΑ ΠΕΡΙΠΤΩΣΗ

Acute acalculous cholecystitis
A consequence or trigger of diabetic ketoacidosis?

ARCHIVES OF HELLENIC MEDICINE 2026, 43(1):132–134
ΑΡΧΕΙΑ ΕΛΛΗΝΙΚΗΣ ΙΑΤΡΙΚΗΣ 2026, 43(1):132–134

A. Aristodimou,
Z. Raptopoulos,
E. Xenofontos

Department of Internal Medicine, State Health Organization Services, General Hospital of Limassol, Limassol, Cyprus

Acute acalculous cholecystitis (AAC) represents 5–10% of all cases of acute cholecystitis and is defined by gallbladder wall thickening, with or without pericholecystic fluid, in the absence of gallstones. We present the case of a 29-year-old man with a medical history of recurrent sinusitis, who presented to the accident and emergency department with a 24-hour history of abdominal pain and fever, alongside a four-month history of polyuria and polydipsia. The patient was diagnosed with severe diabetic ketoacidosis (DKA) with a pH of 6.9, subsequently developing AAC. He was managed conservatively.

Οξεία αλιθιασική χολοκυστίτιδα: Αιτία ή αποτέλεσμα της διαβητικής κετοξέωσης;

Περίληψη στο τέλος του άρθρου

Key words

Acute acalculous cholecystitis
Acute cholecystitis
Computed tomography
Diabetic ketoacidosis
Ultrasound

Submitted 26.11.2024
Accepted 21.12.2024

Acute acalculous cholecystitis (AAC) is defined as inflammation of the gallbladder wall in the absence of gallstones.[1] It accounts for approximately 5–10% of all cases of acute cholecystitis.[2] AAC can be further classified histologically into simple AAC, acute suppurative acalculous cholecystitis, gangrenous acalculous cholecystitis, and AAC leading to gallbladder perforation.[1] AAC is predominantly observed in critically ill patients, such as those admitted to the intensive care unit (ICU) for extended periods, trauma patients, burn patients, or individuals with other severe conditions.[3] Various pathophysiological mechanisms contribute to AAC, depending on its etiology. In cases involving acute ischemia due to shock, the gallbladder artery –a terminal artery– undergoes compensatory constriction to maintain systemic perfusion. This process compromises blood flow to the gallbladder, resulting in ischemia, inflammation, and necrosis.[1]

The diagnosis of AAC is primarily made through ultrasonography (US), which reveals gallbladder wall thickening in the absence of gallstones. The presence of pericholecystic fluid further increases the specificity of the diagnosis.[3] Computed tomography (CT) imaging also plays a crucial role, especially in critically ill patients who may not present with typical symptoms such as right upper quadrant pain or a positive Murphy sign. These patients often undergo abdominal and pelvic CT imaging to investigate alternative diagnoses.[4] Diagnostic criteria for AAC include gallbladder wall thickness >3 mm, gallbladder distension >5 cm, and pericholecystic fluid, all in the absence of gallstones.[5]

Management of AAC typically involves surgical intervention, which remains the golden standard. Treatment options include cholecystectomy or percutaneous cholecystostomy, with or without delayed cholecystectomy following resolution of the acute phase.[6]

CASE PRESENTATION

A 29-year-old male with a history of recurrent sinusitis presented with a 24-hour history of epigastric pain, accompanied by a five-day history of generalized fatigue associated with fever, for which he had been prescribed amoxicillin/clavulanic acid, suspecting sinusitis. He also reported polyuria and polydipsia over the past four months, associated with weight loss. The patient denied any changes in appetite, shortness of breath, cough, chest pain,

diarrhea, joint pain, genital discharge, or rashes. He is a smoker, currently using IQOS.

On admission, the patient was afebrile and hemodynamically stable but exhibited tachypnea and sinus tachycardia on electrocardiogram (ECG). His Glasgow Coma Scale (GCS) score was 15/15. Cardiac examination revealed normal and regular heart sounds with no added murmurs. Lungs were clear with bilateral vesicular breath sounds. Abdominal examination showed a soft and tender epigastric region without rebound tenderness or guarding. Bowel sounds were normal on auscultation. Neurological examination revealed no neck stiffness, and pupils were bilaterally equal and reactive to light.

An arterial blood gas (ABG) analysis revealed severe metabolic acidosis with a pH of 6.9. Blood glucose on ABG was 389 mg/dL, and urine tested positive for ketones.

Additional laboratory findings were significant for leukocytosis (14,740/μL) with a left shift (neutrophils 10,800/μL), hyponatremia (132 mmoL/L), hypokalemia (3.4 mmoL/L), hypochloremia (96 mmoL/L), elevated serum amylase (196 IU/L), hypocalcemia (corrected calcium 7.6 mmoL/L), hypomagnesemia (0.63 mmoL/L), hypophosphatemia (1 mmoL/L), HbA_{1c} 15.2%. Blood and urine cultures were negative, and a chest X-ray revealed no abnormalities.

The patient was admitted to the internal medicine department and treated for severe diabetic ketoacidosis (DKA) per protocol with aggressive fluid resuscitation and an insulin drip, alongside intravenous bicarbonate infusion. However, subsequent ABG testing showed no significant improvement, prompting transfer to the intensive care unit (ICU) for closer monitoring and treatment. While in the ICU, the patient continued to experience epigastric pain radiating to the right upper quadrant (RUQ) with fever, elevated inflammatory markers, and persistently elevated serum amylase. Abdominal US revealed a thickened gallbladder wall (9 mm) without evidence of gallstones, accompanied by mild hepatosplenomegaly. The patient was commenced on intravenous ceftriaxone (2 g once daily) and metronidazole (500 mg three times daily). The surgical team evaluated him and recommended conservative management with follow-up in the outpatient clinic.

The patient's clinical and laboratory parameters improved, and he was discharged on a basal-bolus insulin regimen, oral cefixime (400 mg once daily), and oral metronidazole (400 mg three times daily) for seven days. Discharge instructions included regular blood sugar monitoring and follow-up appointments in the diabetes and general surgery outpatient clinics.

DISCUSSION

Acute acalculous cholecystitis (AAC) is a relatively uncommon cause of acute cholecystitis, accounting for approximately 5–10% of all cases.[2] It is most frequently observed in critically ill patients.[2] The pathophysiology of AAC primarily involves ischemia of the gallbladder, which activates phospholipase A2 and superoxide dismutase, leading to tissue damage.[1] Prolonged ischemia causes hypokinesis of the gallbladder, resulting in bile stasis, increased intraluminal pressure, and further ischemia and necrosis.[1] Additionally, hematogenous spread of gram-negative enteric bacteria is a recognized mechanism in AAC.[1]

Another mechanism discussed in the literature is hyperglycemia-induced gastrointestinal dysmotility (affecting the stomach, intestines, and gallbladder), combined with mesenteric ischemia due to reduced intravascular volume.[7] AAC is associated with a poor prognosis; nearly half of the cases progress to gangrene, and approximately 10% result in gallbladder perforation.[5] In this case, AAC developed in a previously healthy and fit 29-year-old male as a result of severe diabetic ketoacidosis (DKA). Despite his admission to the ICU for only 24 hours, AAC was identified and managed conservatively with positive outcomes.

Management of AAC lacks standardized guidelines. While some studies advocate for surgical intervention, such as cholecystectomy or percutaneous cholecystostomy, others recommend conservative management, reserving surgery for complications like gangrene, empyema, or perforation.[6,8] For example, a case report of AAC in a patient with adult-onset Still's disease highlighted a conservative approach focused on addressing the underlying etiology rather than immediate gallbladder removal.[9] This underscores the importance of tailoring treatment to the patient's clinical presentation and underlying cause.

In the presented case, the patient's condition improved with conservative management, eliminating the need for surgical intervention. However, a follow-up with the surgical team was arranged to monitor disease progression. A study on AAC secondary to viral hepatitis demonstrated that follow-up US revealed a reduction in gallbladder wall thickness following conservative management, emphasizing the utility of imaging in post-treatment evaluations.[8]

ΠΕΡΙΛΗΨΗ

Οξεία αλιθιασική χολοκυστίτιδα: Αιτία ή αποτέλεσμα της διαβητικής κετοξέωσης;

Α. ΑΡΙΣΤΟΔΗΜΟΥ, Ζ. ΡΑΠΤΟΠΟΥΛΟΣ, Ε. ΞΕΝΟΦΩΝΤΟΣ

Τμήμα Εσωτερικής Παθολογίας, Οργανισμός Κρατικών Υπηρεσιών Παροχής Υγείας, Γενικό Νοσοκομείο Λεμεσού, Λεμεσός, Κύπρος

Αρχεία Ελληνική Ιατρικής 2026, 43(1):132–134

Η οξεία αλιθιασική χολοκυστίτιδα αντιπροσωπεύει το 5–10% των περιπτώσεων οξείας χολοκυστίτιδας και χαρακτηρίζεται από πάχυνση του τοιχώματος της χοληδόχου κύστης, με ή χωρίς την παρουσία ελεύθερου υγρού περιχολοκυστικά και σε απουσία λίθων. Το περιστατικό που περιγράφεται αφορά σε έναν άνδρα ηλικίας 29 ετών που προσήλθε στο τμήμα αντιμετώπισης επειγόντων περιστατικών με πολυουρία και πολυδιψία τους τελευταίους 4 μήνες, καθώς και κοιλιακό άλγος και εμπύρετο από 24ώρου. Ο ασθενής διαγνώστηκε με σοβαρή διαβητική κετοξέωση και με οξεία αλιθιασική χολοκυστίτιδα που αντιμετωπίστηκε επιτυχώς συντηρητικά.

Λέξεις ευρετηρίου: Αξονική τομογραφία, Διαβητική κετοξέωση, Οξεία αλιθιασική χολοκυστίτιδα, Οξεία χολοκυστίτιδα, Υπερηχογράφημα

References

1. FU Y, PANG L, DAI W, WU S, KONG J. Advances in the study of acute acalculous cholecystitis: A comprehensive review. *Dig Dis* 2022, 40:468–478
2. JONES MW, FERGUSON T. *Acalculous cholecystitis.* StatPearls, Treasure Island, FL, 2025
3. ADDAS MJ, ALSHAMMARI RM, ALOKAYLI AM, BEDAIWI AK, GHABBAN AM, ALASIRI AM ET AL. Evaluation of acalculous cholecystitis, diagnosis, and management. *Arch Pharm Pract* 2019, 10:17–20
4. PINTO A, REGINELLI A, CAGINI L, COPPOLINO F, STABILE IANORA AA, BRACALE R ET AL. Accuracy of ultrasonography in the diagnosis of acute calculous cholecystitis: Review of literature. *Crit Ultrasound J* 2013, 5(Suppl 1):S11
5. CHANG C, WANG Y, SHI W, XU H, HUANG X, JIAO Y. Is conservative management a safe approach for patients with acute acalculous cholecystitis presenting with an acute abdomen? *Medicine (Baltimore)* 2023, 102:e34662
6. BALMADRID B. Recent advances in management of acalculous cholecystitis. *F1000Res* 2018, 7:F1000 Faculty Rev-1660
7. BELLO CT, GAGO MF, FERNANDES F, OLIVEIRA MM. Abdominal pain in diabetic ketoacidosis: Beyond the obvious. *J Endocrinol Metab* 2018, 8:43–46
8. KAYA S, ESKAZAN AE, AY N, BAYSAL B, BAHADIR MV, ONUR A ET AL. Acute acalculous cholecystitis due to viral hepatitis A. *Case Rep Infect Dis* 2013, 2013:407182
9. VALLIANOU NG, KOUVIDOU C, NAXAKI A, ARISTODIMOU A. Acalculous cholecystitis with multiple organ failure and disseminated intravascular coagulation in a patient with adult onset Still's disease. *Ann Gastroenterol* 2014, 27:289–290

Corresponding author:

A. Aristodimou, Department of Internal Medicine, General Hospital of Limassol, 1 Nikaias street, 4131 Pano Polemidia, Limassol, Cyprus
e-mail: a.aristodimou@gmail.com