EURJIMR | European Journal of Innovative Medical Research

Type CASE REPORT
Published 05.01.2026
DOI 10.65495/eurjimr.2026.7

Successful Conservative Management of Emphysematous Cholecystitis in a Non-Diabetic Elderly Patient with Multiple Comorbidities

Cemalettin Yanık[1], Caner Çulha[2], Emre Boyun[3], Emre Acartürk[4]

[1] Alaca State Hospital, Department of Internal Medicine, Çorum, Türkiye
[2] Taşova State Hospital, Department of Internal Medicine, Amasya, Türkiye
[3] Ayancık State Hospital, Department of Internal Medicine, Sinop, Türkiye
[4] Ondokuz Mayıs University, Faculty of Medicine, Department of Internal Medicine, Samsun, Türkiye

OPEN ACCESS

CORRESPONDENCE
Cemalettin Yanık
cemal_yanik@outlook.com

RECEIVED
18.11.2025
ACCEPTED
26.12.2025
PUBLISHED
05.01.2026

CITATION
Yanık C, Çulha C, Boyun E, Acartürk E. Successful Conservative Management of Emphysematous Cholecystitis in a Non-Diabetic Elderly Patient with Multiple Comorbidities. *Eur J Innov Med Res.* 2026;1(1):22-25. doi:10.65495/eurjimr.2026.7

FINANCIAL SUPPORT
No external funding was received for this study.

CONFLICT OF INTEREST
The authors declare that they have no conflicts of interest related to this study.

ETHICAL APPROVAL
Not applicable.

INFORMED CONSENT
Written informed consent was obtained from the patient.

ACKNOWLEDGEMENTS
None.

PEER REVIEW
Reviewed by at least two peer-reviewers.

ABSTRACT

Background: Emphysematous cholecystitis (EC) is a rare, fulminant variant of acute cholecystitis caused by gas-forming organisms in the setting of gallbladder ischemia and impaired mucosal defenses. It typically affects elderly diabetic men and carries a high mortality rate, particularly when associated with Clostridium perfringens. Although urgent cholecystectomy is considered the standard of care, emerging evidence suggests that early-stage EC may occasionally be managed non-operatively in carefully selected high-risk patients.

Case Presentation: A 79-year-old non-diabetic male with coronary artery disease, prior cerebrovascular accident, acute kidney injury, and hyperlipidemia presented with right upper quadrant pain and nausea. Laboratory studies revealed leukocytosis, markedly elevated CRP, and impaired renal function. Contrast-enhanced CT demonstrated Type I emphysematous cholecystitis with intraluminal gas but no mural involvement, pericholecystic extension, gallstones, or perforation. Due to his high surgical risk, the patient was treated conservatively with IV piperacillin–tazobactam, fluid resuscitation, and close hemodynamic monitoring. On day two, he developed hypotension, oliguria, and rising inflammatory markers, prompting escalation to meropenem. Following antibiotic adjustment, he exhibited continuous clinical and biochemical improvement, with normalization of renal function and resolution of inflammatory markers. Repeat imaging confirmed complete resolution, and the patient was discharged on day ten in stable condition.

Conclusion: This case demonstrates that early-stage emphysematous cholecystitis may be successfully managed with antibiotics alone in non-diabetic patients with significant vascular comorbidities when prompt diagnosis, close monitoring, and appropriate antimicrobial escalation are ensured. Early CT imaging and individualized, stage-based decision-making are essential to avoid progression to gangrene, perforation, or sepsis, particularly in high-risk patients and resource-limited environments.

Keywords: Emphysematous cholecystitis, Acalculous cholecystitis, Conservative management, Acute cholecystitis

Introduction

Emphysematous cholecystitis is a rare but fulminant variant of acute cholecystitis characterized by the presence of gas within the gallbladder lumen, wall, or surrounding tissues.[1,2] Unlike uncomplicated calculous cholecystitis, emphysematous cholecystitis arises from infection with gas-forming organisms—most commonly *Clostridium perfringens, Escherichia coli, Klebsiella spp.,* or anaerobic pathogens—and is driven by a combination of gallbladder ischemia, impaired mucosal defenses, and rapid bacterial proliferation.[1,3–5] Although EC represents only about 1% of acute cholecystitis, mortality ranges from 15%–25%, with even higher rates in fulminant Clostridial infections.[3,6,7]

Classically, emphysematous cholecystitis affects elderly diabetic men with vasculopathy; however, recent reports demonstrate that the condition may occur even in the absence of diabetes when alternative mechanisms—such as diffuse atherosclerosis, immunosuppression, dehydration, or microvascular injury—compromise gallbladder perfusion.[1,2,8–13] Early cross-sectional imaging is critical, as emphysematous cholecystitis may initially mimic uncomplicated cholecystitis, and delayed recognition contributes significantly to its lethality.[2,3,6,7]

Prompt diagnosis is critical, as the condition can evolve rapidly into gangrene, perforation, retroperitoneal gas spread, or fulminant septic shock.[3,8,13,14] Although urgent cholecystectomy remains the standard of care, the expanding body of literature includes rare but compelling examples of successful conservative management in carefully selected patients without peritonitis, perforation, abscess, or hemodynamic instability.[1,10,15,16]

We report a rare case of acalculous, early-stage emphysematous cholecystitis in a non-diabetic elderly male with vascular comorbidities, managed successfully with antibiotics alone in a resource-limited rural hospital, underscoring the importance of early imaging and tailored clinical decision-making.

Case Presentation

A 79-year-old male presented with a two-day history of acute right upper quadrant pain and nausea. His medical history was notable for coronary artery disease, a prior cerebrovascular accident, acute renal failure, and hyperlipidemia. Importantly, he had no history of diabetes mellitus—a key risk factor in most reported cases—nor any previous biliary interventions.

On presentation, he was afebrile, hemodynamically stable, and exhibited no features of systemic sepsis. Physical examination revealed localized right upper quadrant tenderness without guarding or rebound.

Laboratory investigations demonstrated leukocytosis and a markedly elevated C-reactive protein (CRP) of 269 mg/L. Liver enzymes and bilirubin were mildly elevated. Serum creatinine was increased to 1.63 mg/dL, accompanied by reduced urine output, consistent with acute kidney injury.

Contrast-enhanced abdominal CT revealed marked gallbladder wall thickening, intraluminal gas, pericholecystic heterogeneity without pericholecystic air, abscess, or perforation (Figure 1).

Given the absence of peritonitis and the patient's extremely high surgical risk profile, a conservative strategy was selected. Intravenous piperacillin–tazobactam was started, along with isotonic fluids, renal support, and close monitoring.

On hospitalization day two, the patient deteriorated with hypotension, reduced urine output, and a further increase in inflammatory markers (CRP 328 mg/L), raising concern for evolving sepsis. Due to the rural setting, blood or bile cultures could not be obtained, but antimicrobial therapy was escalated to IV meropenem, hydration was intensified, and supportive measures were reinforced. The decision was made as a case-specific

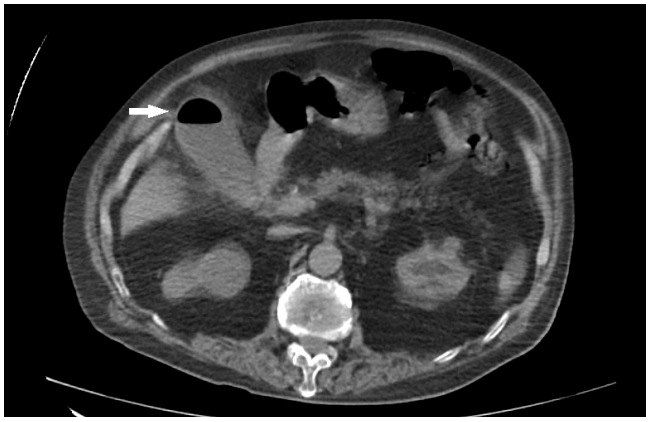

Figure 1. Contrast-enhanced CT of the abdomen. The arrow indicates intraluminal gas within the gallbladder, characteristic of Type I emphysematous cholecystitis, without evidence of wall disruption or perforation.

decision to cover potential resistant anaerobic or Gram-negative pathogens in a deteriorating patient.

Following the antibiotic change, the patient demonstrated continuous clinical improvement: abdominal pain subsided, urine output normalized, and inflammatory markers steadily declined. Renal function and bilirubin levels returned to baseline. Serial ultrasonography confirmed the absence of fluid collections or wall defects. Ursodeoxycholic acid was initiated to support biliary physiology and prevent biliary stasis/sludge during the recovery phase, a strategy selected based on the patient's acalculous presentation and high risk of recurrence.

By day ten, CRP had decreased to 14 mg/L, and serum creatinine normalized to 1.19 mg/dL. The patient was asymptomatic, afebrile, tolerating oral intake, and was discharged after completing the antibiotic regimen. Follow-up imaging demonstrated complete resolution of emphysematous changes.

Informed consent was obtained from the patient for hospitalization, medical treatment, and the anonymous publication of the clinical details and associated imaging for scientific purposes.

Discussion

Emphysematous cholecystitis accounts for approximately 1% of all cases of acute cholecystitis but carries disproportionately high morbidity and mortality due to its rapid progression, vascular compromise, and the virulence of gas-forming organisms.[1–3] Diabetes mellitus remains the most frequently reported predisposing factor; however, the literature consistently emphasizes the contribution of generalized vascular insufficiency, atherosclerotic disease, renal dysfunction, immunosuppression, and transient hemodynamic insults such as dehydration or systemic hypoperfusion.[1,4,12-14,17,18] In this context, EC should be understood as the result of an ischemic–infectious cascade rather than a purely metabolic complication of diabetes.

Radiologically, EC has been categorized according to the distribution of gas within the gallbladder and surrounding structures.[3,19] A commonly used classification divides EC into three types; Type I as the gas is confined to the gallbladder lumen,

type II as the gas as within the gallbladder wall (intramural gas) and type III as the gas is extending into the pericholecystic tissues, biliary ducts, or adjacent spaces.[3,19] This classification carries both diagnostic and therapeutic implications, as Types II and III are more often associated with gangrene, perforation, abscess formation, and systemic sepsis, frequently requiring urgent surgical intervention.[3,19] In our case, contrast-enhanced CT demonstrated intraluminal gas without mural or pericholecystic gas, corresponding to Type I emphysematous cholecystitis. There was no evidence of gallstones, pneumobilia, perforation, or abscess, and no diffuse peritonitis on clinical examination. This early, localized stage of disease likely provided a narrow therapeutic window in which conservative management could be attempted.

Although diabetes is considered the hallmark comorbidity in EC, our patient was non-diabetic, highlighting that diabetic status is neither necessary nor sufficient for the development of this entity. Instead, he had extensive atherosclerotic cardiovascular disease, prior cerebrovascular events, and acute kidney injury, all of which suggest a global microvascular and macrovascular compromise. Ischemic damage to the gallbladder wall is believed to facilitate mucosal breakdown, bacterial translocation, and the proliferation of gas-forming organisms such as *Clostridium perfringens, Escherichia coli, Klebsiella spp.,* and other facultative or obligate anaerobes.[1,3,5] The constellation of advanced age, vascular disease, and renal impairment in this patient is therefore pathophysiologically coherent with current concepts of EC, even in the absence of diabetes or gallstones.[1,3,5,8] This aligns with more recent reports describing EC in elderly non-diabetic patients with significant cardiovascular comorbidity.[3,8-10]

From a diagnostic perspective, computed tomography is widely regarded as the most sensitive modality for detecting even small volumes of gas in the gallbladder lumen and wall.[9,15,20] While ultrasonography may reveal characteristic findings such as echogenic foci with posterior "dirty" shadowing or the "champagne sign," early EC can be subtle and is sometimes missed on initial sonography, with diagnosis made only after CT or repeat imaging.[2,11,15] In our patient, early CT imaging was decisive in confirming Type I EC before the onset of gangrenous changes, perforation, retroperitoneal extension, or biliary sepsis—complications that are well documented and frequently lethal. The ability to differentiate early EC from uncomplicated calculous or acalculous cholecystitis has direct consequences for both prognosis and management strategy.

Traditional management of EC has centered on urgent cholecystectomy, often via an open approach because of severe inflammation and difficulty in identifying Calot's triangle.[1,5,7] However, contemporary practice has broadened the therapeutic spectrum to include emergency cholecystectomy (open or laparoscopic) in operable patients, particularly in Types II–III or when perforation, abscess, or generalized peritonitis is present, percutaneous cholecystostomy as a temporizing or definitive measure in critically ill, high-risk patients, often combined with broad-spectrum intravenous antibiotics, endoscopic transpapillary gallbladder drainage in selected high-risk or perforated cases where percutaneous access is contraindicated or conservative antibiotic therapy alone in rare, carefully selected patients with early-stage, localized disease and no radiologic or clinical evidence of complications.[5,6,8,10,13,15,16,21,22] Our patient was hemodynamically stable at presentation, had Type I intraluminal gas without wall disruption or pericholecystic extension, and was a poor surgical candidate due to age, significant cardiovascular disease, and acute kidney injury. These features supported an initial trial of conservative management.

The inclusion of Ursodeoxycholic acid in our conservative regimen was a case-specific decision aimed at improving biliary physiology and preventing further stasis in a high-risk, non-operative candidate . This approach is supported by recent literature, such as Di Stefano et al, who documented the effectiveness of ursodeoxycholic acid as part of a conservative medical treatment bundle for acute cholecystitis to avoid the need for cholecystectomy in patients where surgical intervention carries significant risk.[23] Furthermore, the use of ursodeoxycholic acid is recognized as a therapeutic option in managing gallbladder sludge and stasis-related disorders when surgical access is limited or contraindicated.[24,25]

In literature, successful non-operative or minimally invasive management strategies for EC generally rely on early, broad-spectrum intravenous antibiotic therapy targeting both aerobic Gram-negative bacteria and anaerobes.[4,6,16] Commonly used regimens include a B-lactam/B-lactamase inhibitor combination (such as piperacillin–tazobactam), a third- or fourth-generation cephalosporin plus metronidazole, or carbapenems (such as meropenem) in cases with clinical deterioration, high suspicion for resistant organisms, or severe sepsis.[4,6,16,26] In our case, we initiated IV piperacillin–tazobactam, consistent with broad coverage recommended for severe biliary infections. On day two, the patient developed hypotension, oliguria, and rising inflammatory markers, suggestive of impending sepsis. Given the rural setting and lack of culture data, antimicrobial therapy was appropriately escalated to IV meropenem, after which the patient's condition progressively improved. This choice was made on a case-by-case basis, but there are examples. Continuous re-evaluation of the need for urgent surgery was a key component of this conservative strategy.

Conclusion

This case highlights that emphysematous cholecystitis can develop in non-diabetic patients with significant vascular comorbidities and may be successfully treated with medical therapy alone when diagnosed early and monitored intensively even in a resource-constrained rural environment. Early CT imaging, rapid antimicrobial escalation, and meticulous hemodynamic follow-up are critical for non-operative success in selected high-risk patients. Although surgery remains the definitive treatment for most cases, individualized, stage-based decision-making can yield excellent outcomes and may prevent unnecessary operative morbidity in vulnerable populations.

References

1. Santoyo Villalba J, Canas Garcia I, Mogollon Gonzalez M. Acute emphysematous cholecystitis due to clostridium perfringens: a rare entity requiring aggressive and early treatment. Letter. Rev Esp Enferm Dig. Jun 2022;114(6):357-358. doi:10.17235/reed.2022.8631/2022

2. Wexler BB, Panebianco NL. The Effervescent Gallbladder: An Emergency Medicine Bedside Ultrasound Diagnosis of Emphysematous Cholecystitis. Cureus. Jul 27 2017;9(7):e1520. e1520. doi:10.7759/cureus.1520

3. Deregowska-Cylke M, Palczewski P, Pacholczyk M, et al. A deceptive case of emphysematous cholecystitis complicated with retroperitoneal gangrene and emphysematous pancreatitis: clinical and computed tomography features. Pol J Radiol. Jan 16 2019;84:e41-e45. doi:10.5114/pjr.2019.82858

4. Kunadia A, Leong MB, Komanduri K, Abdelmasih R, Tarasiuk-Rusek A. Emphysematous Cholecystitis Secondary to Fusobacterium nucleatum. Cureus. Jun 2021;13(6):e15660. e15660. doi:10.7759/cureus.15660

5. Funahashi H, Komori T, Sumita N. Successful treatment of emphysematous cholecystitis by laparoscopic surgery. J Surg Case Rep. Mar 2021;2021(3):rjab080. rjab080. doi:10.1093/jscr/rjab080

6. Hoshi Y, Takeshima K, Matsuoka S, et al. Survival in a Case of Emphysematous Cholecystitis With Sepsis Caused by Clostridium perfringens. Cureus. Nov 2023;15(11):e49705. e49705. doi:10.7759/cureus.49705

7. Slama EM, Bankhead-Kendall B, Robinson RB. Emphysematous Cholecystitis: A Brief Report Demonstrating the Pertinence of Timely Diagnosis. Am Surg. Nov 2020;86(11):1580-1581. doi:10.1177/0003134820940253

8. Misir AP, Vahora I, Unbehaun G, Patel C, Tiesenga F. Perforated Emphysematous Cholecystitis: A Race Against Time. Cureus. Feb 2023;15(2):e35123. doi:10.7759/cureus.35123

9. Sitaridis K, Domi I, Nathanailidou M, et al. Air in gallbladder: Emphysematous cholecystitis or else? Editorial Material. Clin Case Rep. Mar 2023;11(3):e6391. e06391. doi:10.1002/ccr3.6391

10. Vera V, Sharma A, Townson AT, Azeem Q, Malam Y, Mathews J. Emphysematous cholecystitis in a patient with porcelain gallbladder. J Surg Case Rep. Jan 2023;2023(1):rjac600. rjac600. doi:10.1093/jscr/rjac600

11. Yamanoglu A, Bilgin S, Celebi Yamanoglu NG, Topal FE. A Rare Ultrasonographic Finding of Emphysematous Cholecystitis: The Champagne Sign. J Emerg Med. Jun 2021;60(6):e151-e153. doi:10.1016/j.jemermed.2021.01.036

12. Heidenreich D, Kreil S, Hofmann WK, Klein S. Acute spontaneous emphysematous cholecystitis and pancreatitis caused by Clostridium perfringens in a patient with steroid-refractory acute graft versus host disease after allogeneic hamatopoietic cell transplantation. Meeting Abstract. Oncol Res Treat. Sep 2022;45(SUPPL 2):140-140.

13. Sanalp Menekşe T, Menekşe B, Kişi Ö. Acute Emphysematous Cholecystitis Rarely Accompanied by Necrotized Cystic Duct: A Case Report. J Emerg Med Case Rep. Sep 2023;14(3):57-59. doi:10.33706/jemcr.1285478

14. Yoshiyama N, Okada H, Miyake T, et al. Emphysematous cholecystitis during the treatment of heat stroke. Acute Med Surg. Jan-Dec 2020;7(1):e613. e613. doi:10.1002/ams2.613

15. Chae MS, Kravchuk OA. Point of care ultrasound in rapid diagnosis of acute cholangitis and emphysematous cholecystitis: a case report. Int J Emerg Med. Feb 20 2025;18(1):29. 29. doi:10.1186/s12245-025-00823-5

16. Imanzadeh A, Kokabi N, Pourjabbar S, et al. Safety and Efficacy of Percutaneous Cholecystostomy for Emphysematous Cholecystitis. ; Proceedings Paper. J Clin Imaging Sci. Mar 27 2020;10:9. 9. doi:10.25259/JCIS_145_2019

17. Kubota Y, Ishioka H, Harada S, et al. Septic shock with emphysematous cholecystitis and disseminated infection caused by hypervirulent Klebsiella pneumoniae capsular genotype K2-ST65 in a Japanese man with diabetes mellitus: A case report. J Infect Chemother. Feb 2021;27(2):350-353. doi:10.1016/j.jiac.2020.09.017

18. Montelongo-Rodriguez FA, Robles-Torres JI, Garcia-Saucedo J, Ruiz-Galindo E, Pacheco-Molina C, Gomez-Guerra LS. Emphysematous Pyelonephritis and Emphysematous Cholecystitis: A Result of Uncontrolled Type 2 Diabetes. Ochsner J. Fall 2021;21(3):316-318. doi:10.31486/toj.20.0126

19. Safwan M, Penny SM. Emphysematous Cholecystitis: A Deadly Twist to a Common Disease. Journal of Diagnostic Medical Sonography. 2016;32(3):131-137. doi:10.1177/8756479316631535

20. Yamaguchi T. Detecting emphysematous cholecystitis using plain radiograph. Editorial Material. Erciyes Medical Journal. 2023 2022;45(2):215-216. doi:10.14744/etd.2022.99897

21. Iino C, Shimoyama T, Igarashi T, et al. Perforated emphysematous cholecystitis managed by endoscopic transpapillary gallbladder drainage. Clin J Gastroenterol. Aug 2017;10(4):388-391. doi:10.1007/s12328-017-0746-z

22. Kelly S, Shah K, Motizada J, Soggiu F, Sheth H. Laparoscopic treatment of an unruptured cystic artery pseudo-aneurysm in the presence of calculous emphysematous cholecystitis. J Surg Case Rep. Jan 2022;2022(1):rjab625. rjab625. doi:10.1093/jscr/rjab625

23. Di Stefano M, Colombo C, De Leo S, et al. High Prevalence and Conservative Management of Acute Cholecystitis during Lenvatinib for Advanced Thyroid Cancer. Eur Thyroid J. 2021;10(4):314-322. doi:10.1159/000510369

24. Pineda O, Maydón HG, Amado M, et al. A Prospective Study of the Conservative Management of Asymptomatic Preoperative and Postoperative Gallbladder Disease in Bariatric Surgery. Obes Surg. 2017;27(1):148-153. doi:10.1007/s11695-016-2264-3

25. Çakar S, Eren G, Erdur CB, et al. Cholelithiasis in Infants: Risk Factors, Management, and the Role of Ursodeoxycholic Acid. Children (Basel). 2024;11(12):1553. Published 2024 Dec 21. doi:10.3390/children11121553

26. Tohya M, Otsuka T, Yoshimoto J, Ishizaki Y, Kirikae T, Watanabe S. Case report: Whole genome sequence of Clostridium perfringens JUM001 causing acute emphysematous cholecystitis. Front Microbiol. Nov 17 2022;13:1066880. 1066880. doi:10.3389/fmicb.2022.1066880