Clinical Assessment, Early Recognition, and Interprofessional Care in Acute Cholecystitis for Emergency and Nursing Practice-An Updated Review

Hamzah Muidh Ahmed Alribi[1], Abdullah Hassan Abdullah Alafifi[2], Maher Ayidh Aljabri[3], Khalid Obid Alruki[4], Ayyashi Dahnan Jubran I[5], Yahya Ali A. Majrashi[6], Khaled Yahya Hassan Khabrani[7], Ibrahim Hussain A. Alanazi[8], Fadhel Ghazi K. Aljoudi[9], Tawfiq Saeed M. Saqer[10]

Abstract

Background: Acute cholecystitis is an inflammatory condition of the gallbladder most commonly resulting from obstruction of the cystic duct by gallstones, leading to bile stasis, increased intraluminal pressure, ischemia, and infection. It represents a frequent cause of acute abdominal pain and surgical admission, particularly among adults with metabolic and hormonal risk factors. The condition may also occur without gallstones in critically ill patients due to impaired gallbladder emptying and systemic illness.

Aim and Methods: The aim of this review is to provide a comprehensive overview of acute cholecystitis with emphasis on clinical assessment, early recognition, and interprofessional management relevant to emergency and nursing practice. A structured narrative review approach was used to synthesize evidence from established clinical literature addressing epidemiology, pathophysiology, diagnosis, complications, and treatment strategies.

Results: Findings indicate that diagnosis relies on integration of clinical presentation, laboratory abnormalities such as leukocytosis and liver enzyme elevation, and imaging modalities including ultrasound, computed tomography, and hepatobiliary scintigraphy. Early laparoscopic cholecystectomy remains the definitive treatment and is associated with reduced morbidity and mortality when performed within 72 hours of symptom onset. Conservative management and percutaneous interventions are reserved for high-risk or unstable patients. Delayed or untreated disease is associated with severe complications, including gallbladder perforation, abscess formation, and sepsis.

Conclusion: In conclusion, acute cholecystitis requires timely diagnosis and coordinated multidisciplinary management to optimize outcomes. Early surgical intervention significantly improves prognosis and reduces complications.

[1]Ministry of National Guard, Riyadh, Saudi Arabia
[2]Ministry of National Guard Health Affairs (MNGHA), Saudi Arabia
[3]Drug and Psychotropic Substances Detection Committee, Madinah, Ministry of National Guard, Saudi Arabia
[4]Health Affairs, Ministry of National Guard, Riyadh, Saudi Arabia
[5]Ministry of National Guard Health Affairs (MNGHA), Saudi Arabia
[6]Ministry of National Guard Health Affairs (MNGHA), Riyadh, Saudi Arabia
[7]Ministry of National Guard Health Affairs (MNGHA), Riyadh, Saudi Arabia
[8]Ministry of National Guard Health Affairs (MNGHA), Riyadh, Saudi Arabia
[9]Ministry of National Guard Health Affairs (MNGHA), Riyadh, Saudi Arabia
[10]Ministry of National Guard Health Affairs (MNGHA), Riyadh, Saudi Arabia

Keywords: acute cholecystitis, gallstones, laparoscopic cholecystectomy, biliary inflammation, emergency care, interprofessional management

Introduction

Acute cholecystitis is an acute inflammatory condition of the gallbladder that most commonly results from obstruction of the cystic duct by gallstones. This obstruction leads to bile stasis, increased intraluminal pressure, and subsequent inflammatory changes within the gallbladder wall. In some clinical situations, the condition develops in the absence of gallstones, particularly in critically ill patients, where impaired gallbladder emptying secondary to cholestasis, prolonged fasting, or systemic illness contributes to inflammation and ischemic injury. These pathophysiological mechanisms highlight the multifactorial nature of the disease process and its association with both mechanical obstruction and systemic physiological stress.[1] Acute cholecystitis may present as part of a broader spectrum of gallbladder disease that includes both acute and chronic forms. The acute form is characterized by sudden onset inflammation with distinct clinical manifestations, whereas chronic cholecystitis typically results from repeated episodes of inflammation leading to gallbladder wall fibrosis and functional impairment. Although both males and females can be affected, epidemiological data consistently show a higher prevalence in females, which is often linked to hormonal influences, pregnancy, and metabolic factors that increase gallstone formation risk.

Clinically, acute cholecystitis presents with a constellation of symptoms that may include right upper quadrant abdominal pain, fever, nausea, and vomiting. However, the diagnosis can be challenging due to symptom overlap with several other conditions. Differential diagnoses such as peptic ulcer disease, irritable bowel syndrome, pancreatitis, and even cardiac ischemia can mimic its presentation, particularly in older patients or those with atypical symptoms.[2][3][4] This overlap necessitates careful clinical evaluation supported by laboratory testing and imaging studies to establish an accurate diagnosis and guide timely management. Early recognition and appropriate treatment are essential to prevent complications such as gallbladder perforation, empyema, or systemic infection. Management strategies vary depending on disease severity and patient stability, ranging from conservative medical therapy with antibiotics and supportive care to definitive surgical intervention, most commonly laparoscopic cholecystectomy. This dual approach reflects the importance of individualized care in optimizing patient outcomes in acute gallbladder inflammation.

Etiology

Acute cholecystitis most commonly arises from obstruction of the cystic duct, which disrupts normal bile flow and initiates a cascade of inflammatory and ischemic changes within the gallbladder. Under physiological conditions, bile is produced continuously by the liver and transported through the hepatic ducts into the gallbladder via the cystic duct. The gallbladder functions as a reservoir, concentrating bile and releasing it into the duodenum through the common bile duct and ampulla of Vater. This process is tightly regulated by cholecystokinin (CCK), a hormone released after ingestion of fatty meals that stimulates gallbladder contraction and coordinated bile delivery to support lipid digestion and absorption.[5] Pathological disruption of this system occurs when bile homeostasis is altered. Factors such as bile stasis, increased hepatic secretion of cholesterol and lipids, impaired gallbladder emptying, and abnormal nucleation of cholesterol crystals contribute to gallstone formation. Over time, these changes promote supersaturation and precipitation of bile constituents, leading to stone development. When a gallstone becomes lodged in the cystic duct, bile outflow is obstructed, resulting in increased intraluminal pressure within the gallbladder. This condition initially presents as biliary colic, characterized by episodic pain in the right upper quadrant or epigastric region. Persistent obstruction leads to progression into acute calculous cholecystitis. This occurs when inflammation becomes sustained, typically when symptoms persist beyond six hours. Continued bile retention causes gallbladder distention, venous congestion, and compromised lymphatic drainage, all of which contribute to progressive inflammatory injury. The majority of cases, approximately 95%, are associated with gallstones, confirming their central role in disease pathogenesis.[8]

In contrast, acute acalculous cholecystitis occurs in the absence of gallstones and is more commonly seen in critically ill patients. It is frequently associated with conditions such as sepsis, trauma, burns, or prolonged dependence on total parenteral nutrition (TPN).[6][7] In these cases, gallbladder stasis and ischemia play a dominant role rather than mechanical obstruction. Reduced perfusion and bile stasis promote mucosal injury and inflammation, even without calculi. Regardless of etiology, sustained obstruction or stasis increases intraluminal pressure, which in turn elevates transmural pressure and impairs microvascular perfusion. This ischemic environment predisposes the gallbladder wall to necrosis and, if untreated, gangrene. Gangrenous transformation significantly increases susceptibility to secondary infection, particularly by gas-forming organisms, leading to emphysematous cholecystitis. These advanced stages carry a high risk of perforation, which can result in biliary peritonitis and severe systemic infection, markedly increasing morbidity and mortality. Importantly, while gallstones are common in the general population, most remain asymptomatic. Evidence indicates that only a minority, approximately 20% over 20 years, progress to symptomatic disease, which supports conservative management in asymptomatic individuals without indication for prophylactic surgery.[9]

Epidemiology

Acute cholecystitis shows a clear epidemiological pattern linked to demographic, metabolic, and genetic risk factors that influence gallstone formation and subsequent biliary obstruction. The condition is more frequently observed in women, particularly during reproductive age, suggesting a strong hormonal influence on cholesterol metabolism and bile composition. Elevated estrogen levels increase cholesterol saturation in bile, while progesterone reduces gallbladder motility, both contributing to stone formation and increasing susceptibility to gallbladder inflammation. Obesity represents another major risk factor. Excess body weight alters lipid metabolism and increases cholesterol secretion into bile, which promotes supersaturation and crystal formation. Patients in their fourth decade of life are particularly affected, reflecting cumulative exposure to metabolic risk factors over time. Rapid weight loss, especially following bariatric surgery or restrictive diets, also increases risk due to enhanced cholesterol mobilization and gallbladder stasis. Similarly, acute systemic illnesses can impair gallbladder emptying, increasing the likelihood of bile retention and inflammation. Pregnancy is an additional high-risk state due to hormonal changes that reduce gallbladder contractility and alter bile composition. These physiological adaptations, while normal during pregnancy, create conditions favorable for sludge formation and gallstone development. Genetic predisposition also plays an important role in disease epidemiology. A family history of gallstones increases individual susceptibility, indicating inherited factors that influence cholesterol metabolism and biliary physiology. This genetic contribution interacts with environmental and metabolic factors to determine overall risk. Certain hematological disorders further contribute to disease development. Conditions associated with increased red blood cell breakdown, such as sickle cell disease, lead to excess bilirubin production. This excess bilirubin precipitates in bile, forming pigment stones rather than cholesterol stones. Patients with such conditions have a significantly increased risk of developing gallstones and subsequent acute cholecystitis due to chronic biliary obstruction and inflammation. Overall, the epidemiology of acute cholecystitis reflects a multifactorial process involving hormonal, metabolic, genetic, and hematological influences that collectively determine disease susceptibility and clinical distribution across populations.

Pathophysiology

Acute cholecystitis develops through a sequence of mechanical obstruction, bile stasis, vascular compromise, and inflammatory injury. The initiating event is most often occlusion of the cystic duct or failure of effective gallbladder emptying. When bile cannot drain properly, it accumulates within the gallbladder lumen, leading to progressive distension and increased intraluminal pressure. This rising pressure compresses the gallbladder wall microcirculation, resulting in reduced venous and lymphatic outflow and subsequent ischemia of the mucosal and muscular layers. Ischemic injury triggers an inflammatory cascade characterized by mucosal damage, edema, and leukocyte infiltration. At the same time, bile stasis promotes bacterial proliferation, particularly enteric organisms that ascend from the duodenum. The combination of chemical irritation from concentrated bile and bacterial infection amplifies local inflammation and accelerates tissue injury. If this process persists, it can lead to necrosis of the gallbladder wall, gangrene, and eventual perforation, which may result in biliary peritonitis, systemic infection, and sepsis. Gallstones play a central role in most cases. These stones are primarily composed of cholesterol or bilirubinate and form when bile composition becomes imbalanced. Cholesterol stones develop when bile becomes supersaturated with cholesterol, often due to metabolic abnormalities that increase hepatic cholesterol secretion. Pigment stones are more commonly associated with conditions that increase bilirubin turnover, such as hemolytic disorders. In diseases like sickle cell disease, chronic hemolysis elevates unconjugated bilirubin levels, increasing the likelihood of pigment stone formation and subsequent biliary obstruction.[11]

Other metabolic and endocrine conditions also contribute to gallstone formation. Hyperparathyroidism, for example, leads to elevated serum calcium levels, which can promote calcium-based stone formation within the biliary system. Pregnancy represents another important risk state due to elevated progesterone levels, which reduce gallbladder contractility and delay emptying, thereby encouraging bile stasis and stone formation. Mechanical obstruction from external sources, such as neoplasms or biliary strictures, can further disrupt bile flow. Persistent obstruction maintains a stagnant biliary environment, promoting crystallization and stone growth while simultaneously increasing the risk of inflammatory activation. Overall, the pathophysiology of acute cholecystitis reflects a dynamic interaction between obstruction, bile composition abnormalities, infection, and ischemic injury, all converging to produce progressive gallbladder inflammation and potential systemic complications.

Histopathology

Acute cholecystitis demonstrates characteristic histopathological changes that evolve with disease progression and reflect the underlying inflammatory and ischemic processes affecting the gallbladder wall. In the early stage of the disease, microscopic examination typically reveals marked vascular congestion within the gallbladder wall, accompanied by interstitial edema. These changes are primarily the result of increased intraluminal pressure following cystic duct obstruction, which impairs venous and lymphatic drainage. The mucosa may show early signs of epithelial injury, while inflammatory cell infiltration begins to develop. As the condition progresses, the inflammatory response becomes more pronounced. Chronic inflammatory cells, including lymphocytes and macrophages, may be observed within the gallbladder wall, indicating sustained tissue injury. Fibrotic changes can also develop over time, reflecting ongoing repair and remodeling processes in response to persistent inflammation. This stage may show thickening of

the gallbladder wall and partial loss of normal mucosal architecture. In more severe or advanced cases, the histopathological findings may progress to gangrenous cholecystitis. This stage is characterized by extensive tissue necrosis, hemorrhage, and destruction of the gallbladder wall. The loss of vascular supply due to sustained ischemia leads to full-thickness necrosis, significantly increasing the risk of perforation. In cases where perforation occurs, histology may reveal rupture of the gallbladder wall with surrounding inflammatory exudate and possible involvement of adjacent hepatic or peritoneal structures. Overall, the histopathological spectrum of acute cholecystitis reflects a continuum from early vascular congestion and edema to chronic inflammation, fibrosis, and ultimately necrosis and gangrene in severe untreated cases [11].

History and Physical

The clinical presentation of Acute cholecystitis and chronic cholecystitis reflects the degree and duration of gallbladder inflammation. A thorough history and focused physical examination are essential for distinguishing between acute and chronic disease and for differentiating gallbladder pathology from other gastrointestinal, hepatobiliary, and cardiovascular disorders. Chronic cholecystitis typically develops gradually and is characterized by recurrent episodes of right upper quadrant abdominal pain that progressively worsen over time. Patients frequently report associated symptoms such as abdominal bloating, nausea, vomiting, dyspepsia, and intolerance to certain foods, particularly fatty, greasy, or spicy meals. Pain often occurs after eating and may persist for several hours before subsiding. In many cases, discomfort radiates to the right shoulder, scapular region, or mid-back due to shared neural pathways between the gallbladder and adjacent structures. Because symptoms may be intermittent and relatively mild during the early stages, diagnosis is often delayed, with some individuals experiencing recurrent episodes for years before definitive evaluation and treatment. Acute cholecystitis presents with a similar symptom profile but is generally more severe and persistent. Patients commonly experience sudden onset of intense right upper quadrant pain, often following the consumption of a high-fat meal that stimulates gallbladder contraction against an obstructed cystic duct. The pain is typically continuous rather than episodic and may be accompanied by fever, nausea, vomiting, anorexia, and general malaise. The severity of symptoms frequently prompts urgent medical evaluation. Physical examination findings are often highly suggestive of acute gallbladder inflammation. Localized tenderness over the right upper quadrant is a common finding, and guarding may be present in more severe cases. One of the most characteristic clinical signs is Murphy's sign, which occurs when deep palpation of the right upper quadrant during inspiration causes sudden cessation of inspiration due to pain. This finding reflects direct contact between the inflamed gallbladder and the examiner's hand and is considered a classic indicator of acute cholecystitis. Because upper abdominal pain may mimic other conditions, including peptic ulcer disease, pancreatitis, hepatitis, and even cardiac ischemia, careful clinical assessment is essential. The overlap of symptoms with cardiovascular disorders is particularly important, as some patients may initially present symptoms suggestive of cardiac disease. Consequently, comprehensive history taking and physical examination remain fundamental components of accurate diagnosis and appropriate management [11][12][13].

Evaluation

The diagnosis of Acute cholecystitis requires integration of clinical findings with laboratory and imaging studies to ensure accurate identification and exclusion of other abdominal pathologies. Clinical assessment remains the cornerstone of evaluation, but laboratory investigations provide important supportive evidence and help guide further diagnostic steps. Basic laboratory tests include a complete blood count (CBC) and a comprehensive metabolic panel (CMP). In chronic cholecystitis, these results may remain within normal ranges, reflecting the intermittent or low-grade nature of inflammation. In acute cases, however, leukocytosis is commonly observed, indicating an active inflammatory or infectious process. Liver function tests may also show elevated transaminases and alkaline phosphatase levels. The presence of hyperbilirubinemia raises clinical suspicion for common bile duct obstruction or more extensive biliary involvement. Despite these trends, laboratory findings can occasionally be normal even in clinically significant disease, which highlights the importance of correlating results with imaging and clinical presentation. Additional laboratory evaluation includes measurement of serum amylase and lipase levels to exclude pancreatitis, which can present with similar epigastric or right upper quadrant pain. Differentiating between these conditions is essential because management strategies differ significantly. Imaging plays a central role in confirming the diagnosis. In emergency settings, computed tomography (CT) is often used as an initial imaging modality due to its ability to rapidly evaluate abdominal structures. CT findings may include gallbladder distention, wall thickening, pericholecystic fluid, visible gallstones, and surrounding inflammatory changes. However, abdominal ultrasound remains the preferred first-line imaging technique due to its high sensitivity for gallstones and lack of radiation exposure. Ultrasound findings consistent with acute cholecystitis include gallbladder wall thickening greater than 3 mm, presence of gallstones, pericholecystic fluid, and gallbladder distention. When ultrasound and CT results are inconclusive, a hepatobiliary iminodiacetic acid (HIDA) scan provides functional assessment of gallbladder activity. Failure of the gallbladder to visualize radiotracer uptake indicates cystic duct obstruction and strongly supports the diagnosis. In cases without gallstones, the administration of cholecystokinin (CCK) during HIDA scanning can evaluate gallbladder contractility. A reduced ejection fraction below 35% suggests biliary dyskinesia or functional

gallbladder disorder. Overall, accurate evaluation depends on combining clinical suspicion with targeted laboratory testing and stepwise imaging to confirm diagnosis and guide appropriate management [14][15].

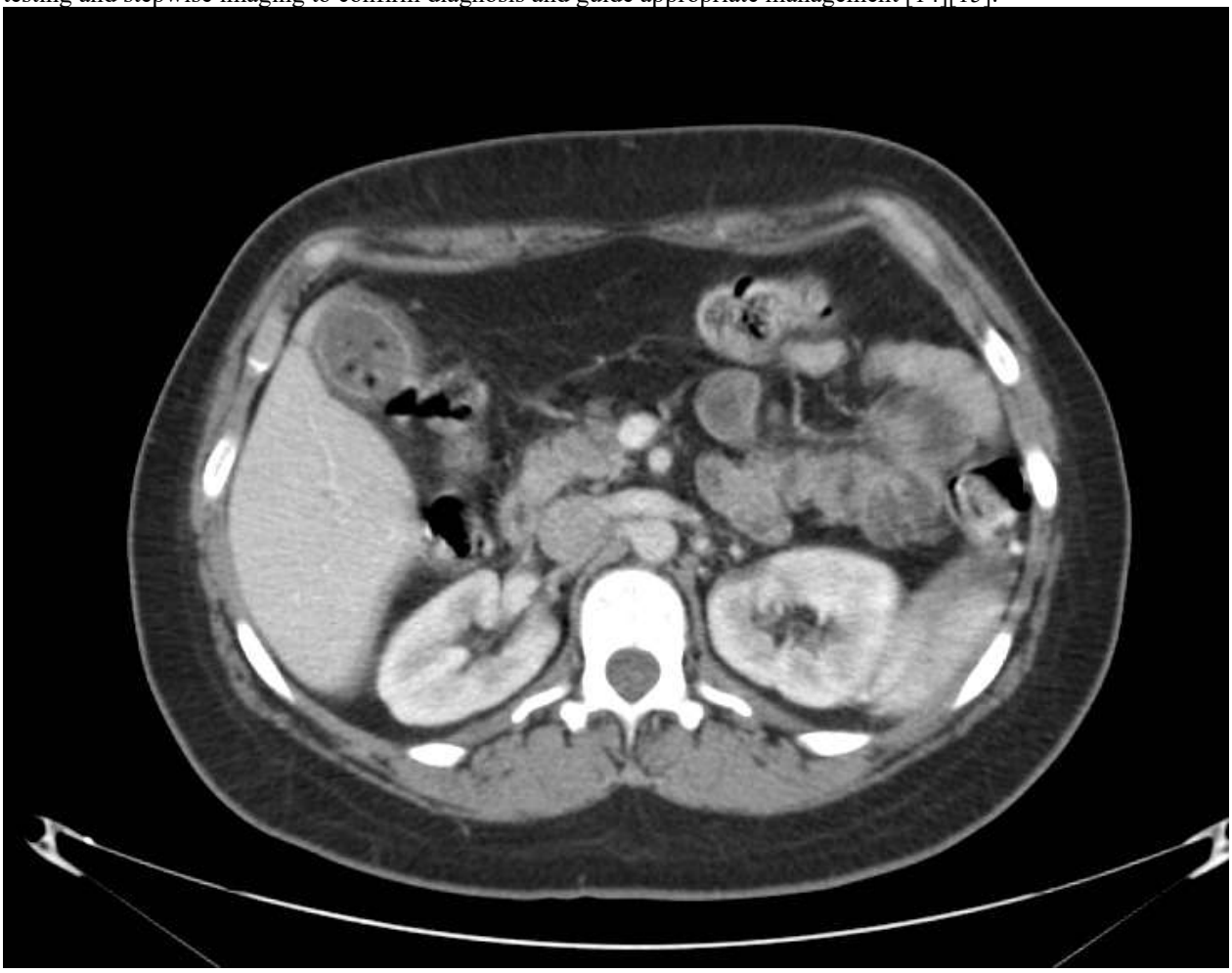

Fig. 1: Acute Cholecystitis.

Treatment / Management

The management of Acute cholecystitis is determined by disease severity, patient comorbidities, and overall surgical risk, with treatment strategies broadly categorized into surgical and non-surgical approaches. Initial management in all patients typically begins with stabilization measures, including intravenous fluid resuscitation to correct dehydration and electrolyte imbalance, alongside early administration of broad-spectrum antibiotics targeting gram-negative organisms and anaerobic bacteria. This early medical optimization is essential to control infection, reduce systemic inflammatory response, and prepare patients for definitive intervention. For patients who are suitable surgical candidates, early laparoscopic cholecystectomy during the index hospitalization represents the standard of care. This approach is strongly supported by clinical evidence demonstrating reduced postoperative complications, shorter hospital stays, and lower overall morbidity and mortality when compared with delayed surgical intervention.[16][17] Early removal of the inflamed gallbladder also prevents recurrence and reduces the risk of complications such as perforation or abscess formation. In circumstances where laparoscopic access is not feasible due to technical limitations, severe inflammation, or patient-related factors, open cholecystectomy remains an accepted alternative. Technological advancements have introduced robotic-assisted cholecystectomy as an additional surgical option in selected centers. Although earlier iterations of robotic systems were associated with concerns regarding bile duct injury, more recent platforms have demonstrated improved precision and enhanced safety profiles. Current evidence suggests a reduction in conversion rates to open surgery and a lower incidence of major complications when compared with conventional laparoscopic techniques, particularly in experienced surgical hands.[18][19][20]

Non-surgical management is reserved primarily for patients with chronic or mild disease who are poor surgical candidates or those with significant operative risk. Conservative measures include dietary modification, particularly adherence to a low-fat diet, which may reduce symptom frequency; however, clinical outcomes remain variable. Pharmacological therapy with ursodeoxycholic acid has been utilized in select cases for gallstone dissolution, although its overall efficacy is limited and not consistently reliable in achieving complete stone resolution.[21][22][4] In critically ill or high-risk patients who are unable to undergo surgery, percutaneous cholecystostomy performed under interventional radiology guidance serves as an important alternative. This procedure may function as a definitive

treatment in frail or elderly individuals or as a temporary bridge to delayed elective cholecystectomy, typically performed within four to eight weeks following stabilization.[23] In patients managed conservatively, outpatient imaging studies may be conducted to evaluate cystic duct patency prior to drain removal; however, recurrence rates of acute cholecystitis after tube removal can reach approximately 47%, highlighting the limitations of this approach.[24] Additional minimally invasive options include endoscopic interventions such as cystic duct stenting and transduodenal gallbladder drainage. These techniques offer promising alternatives for patients who are unfit for surgery, although their availability remains limited and long-term outcome data are still evolving. Consequently, individualized treatment planning based on patient risk stratification and multidisciplinary evaluation remains essential for optimal clinical outcomes.

Differential Diagnosis

Accurate identification of Acute cholecystitis requires careful exclusion of multiple gastrointestinal, hepatobiliary, and systemic conditions that present with overlapping clinical features. Misinterpretation of symptoms can delay treatment and increase the risk of complications, making structured differential assessment essential in clinical practice. Biliary colic represents a transient obstruction of the cystic duct by gallstones without sustained inflammation. Choledocholithiasis involves gallstones within the common bile duct and may present with jaundice and cholestatic liver enzyme patterns. Cholangitis is characterized by biliary obstruction with superimposed infection and typically presents with systemic toxicity, fever, and jaundice. Pancreatitis should also be considered, particularly when epigastric pain radiates to the back and serum lipase is elevated. Hepatitis may mimic gallbladder disease through right upper quadrant discomfort and abnormal liver enzymes, but it is usually associated with diffuse hepatic inflammation rather than localized gallbladder pathology. Upper gastrointestinal disorders such as gastritis, hiatal hernia, and peptic ulcer disease can present with epigastric pain and dyspeptic symptoms that overlap with gallbladder inflammation. Appendicitis, especially in atypical presentations, may also produce abdominal pain that requires differentiation based on localization and progression. Mesenteric ischemia must be considered in high-risk patients presenting severe abdominal pain disproportionate to physical findings. Small bowel obstruction can similarly mimic biliary disease through abdominal distension, vomiting, and pain patterns. Clinical differentiation relies on integrating symptom progression, laboratory abnormalities, and imaging findings to avoid diagnostic error and ensure appropriate early management [23][24][25][26].

Prognosis

The outcome of Acute cholecystitis is highly dependent on the timing of diagnosis and intervention. Untreated disease carries a significant risk of severe complications, particularly in older adults and patients with comorbid conditions. Gallbladder perforation represents a critical complication associated with mortality rates reaching approximately 30%, reflecting the severity of uncontrolled inflammation and infection.[27] Early surgical management significantly improves clinical outcomes. Patients who undergo laparoscopic cholecystectomy within 72 hours of symptom onset demonstrate substantially reduced morbidity and mortality, with reported 30-day morbidity around 6.6% and mortality approximately 1.1%.[17] Early intervention limits progression to gangrene, perforation, and systemic infection, thereby improving overall survival and reducing hospital length of stay. Prognosis is generally favorable in uncomplicated cases when timely treatment is provided, whereas delayed management is strongly associated with increased complication rates and poorer outcomes.

Complications

Complications of Acute cholecystitis arise from progressive inflammation, infection, and procedural risks. Intraabdominal abscess formation may occur following localized perforation or uncontrolled infection. Gallbladder perforation represents a severe complication that can lead to generalized peritonitis and systemic sepsis. Cholecystoenteric fistulas may develop due to chronic inflammation and erosion into adjacent bowel structures. Other complications include biloma formation resulting from bile leakage, bile duct injury during surgical intervention, and hepatic injury due to inflammatory extension. Bowel injury may occur as a procedural complication during cholecystectomy. Postoperative infection remains a concern, particularly in complicated or delayed cases. Retained common bile duct stones can result in persistent biliary obstruction and recurrent symptoms. Hemorrhage and bleeding are additional risks associated with surgical or interventional procedures. These complications highlight the importance of early diagnosis, careful surgical technique, and close postoperative monitoring to reduce morbidity and prevent long-term sequelae [24].

Conclusion

Acute cholecystitis remains a common surgical emergency with significant potential for morbidity when diagnosis or treatment is delayed. The condition arises mainly from cystic duct obstruction, leading to progressive inflammation, ischemia, and possible gallbladder necrosis. Clinical presentation varies from mild biliary symptoms to severe systemic infection, requiring careful assessment to avoid misdiagnosis. Imaging and laboratory investigations play a key role in confirming the diagnosis and guiding management decisions. Early recognition and timely intervention significantly improve outcomes. Laparoscopic cholecystectomy performed within the early phase of admission

remains the definitive treatment and is associated with lower complication rates and improved survival. Conservative and interventional approaches are reserved for high-risk or unstable patients. Multidisciplinary collaboration between emergency physicians, surgeons, radiologists, nurses, and pharmacists is essential to optimize patient care. Complications such as perforation, abscess formation, and sepsis highlight the importance of rapid clinical decision-making. With appropriate management, prognosis is generally favorable, emphasizing the value of early diagnosis, structured evaluation, and evidence-based treatment pathways in reducing disease burden.

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(4):468-478.
2. Burmeister G, Hinz S, Schafmayer C. [Acute Cholecystitis]. Zentralbl Chir. 2018 Aug;143(4):392-399.
3. Walsh K, Goutos I, Dheansa B. Acute Acalculous Cholecystitis in Burns: A Review. J Burn Care Res. 2018 Aug 17;39(5):724-728.
4. Kohga A, Suzuki K, Okumura T, Yamashita K, Isogaki J, Kawabe A, Kimura T. Is postponed laparoscopic cholecystectomy justified for acute cholecystitis appearing early after onset? Asian J Endosc Surg. 2019 Jan;12(1):69-73.
5. Ahmed M. Functional, Diagnostic and Therapeutic Aspects of Bile. Clin Exp Gastroenterol. 2022;15:105-120.
6. Yun SP, Seo HI. Clinical aspects of bile culture in patients undergoing laparoscopic cholecystectomy. Medicine (Baltimore). 2018 Jun;97(26):e11234.
7. Wilkins T, Agabin E, Varghese J, Talukder A. Gallbladder Dysfunction: Cholecystitis, Choledocholithiasis, Cholangitis, and Biliary Dyskinesia. Prim Care. 2017 Dec;44(4):575-597.
8. Halpin V. Acute cholecystitis. BMJ Clin Evid. 2014 Aug 20;2014
9. Behari A, Kapoor VK. Asymptomatic Gallstones (AsGS) - To Treat or Not to? Indian J Surg. 2012 Feb;74(1):4-12.
10. Wang AJ, Wang TE, Lin CC, Lin SC, Shih SC. Clinical predictors of severe gallbladder complications in acute acalculous cholecystitis. World J Gastroenterol. 2003 Dec;9(12):2821-3.
11. Tierney S, Nakeeb A, Wong O, Lipsett PA, Sostre S, Pitt HA, Lillemoe KD. Progesterone alters biliary flow dynamics. Ann Surg. 1999 Feb;229(2):205-9.
12. Apolo Romero EX, Gálvez Salazar PF, Estrada Chandi JA, González Andrade F, Molina Proaño GA, Mesías Andrade FC, Cadena Baquero JC. Gallbladder duplication and cholecystitis. J Surg Case Rep. 2018 Jul;2018(7):rjy158.
13. Sureka B, Rastogi A, Mukund A, Thapar S, Bhadoria AS, Chattopadhyay TK. Gangrenous cholecystitis: Analysis of imaging findings in histopathologically confirmed cases. Indian J Radiol Imaging. 2018 Jan-Mar;28(1):49-54.
14. Tootian Tourghabe J, Arabikhan HR, Alamdaran A, Zamani Moghadam H. Emergency Medicine Resident versus Radiologist in Detecting the Ultrasonographic Signs of Acute Cholecystitis; a Diagnostic Accuracy Study. Emerg (Tehran). 2018;6(1):e19.
15. Joshi G, Crawford KA, Hanna TN, Herr KD, Dahiya N, Menias CO. US of Right Upper Quadrant Pain in the Emergency Department: Diagnosing beyond Gallbladder and Biliary Disease. Radiographics. 2018 May-Jun;38(3):766-793.
16. Brooks KR, Scarborough JE, Vaslef SN, Shapiro ML. No need to wait: an analysis of the timing of cholecystectomy during admission for acute cholecystitis using the American College of Surgeons National Surgical Quality Improvement Program database. J Trauma Acute Care Surg. 2013 Jan;74(1):167-73; 173-4.
17. Fugazzola P, Cobianchi L, Di Martino M, Tomasoni M, Dal Mas F, Abu-Zidan FM, Agnoletti V, Ceresoli M, Coccolini F, Di Saverio S, Dominioni T, Farè CN, Frassini S, Gambini G, Leppäniemi A, Maestri M, Martín-Pérez E, Moore EE, Musella V, Peitzman AB, de la Hoz Rodríguez Á, Sargenti B, Sartelli M, Viganò J, Anderloni A, Biffl W, Catena F, Ansaloni L., S.P.Ri.M.A.C.C. Collaborative Group. Prediction of morbidity and mortality after early cholecystectomy for acute calculous cholecystitis: results of the S.P.Ri.M.A.C.C. study. World J Emerg Surg. 2023 Mar 18;18(1):20.
18. Lunardi N, Abou-Zamzam A, Florecki KL, Chidambaram S, Shih IF, Kent AJ, Joseph B, Byrne JP, Sakran JV. Robotic Technology in Emergency General Surgery Cases in the Era of Minimally Invasive Surgery. JAMA Surg. 2024 May 01;159(5):493-499.
19. Kalata S, Thumma JR, Norton EC, Dimick JB, Sheetz KH. Comparative Safety of Robotic-Assisted vs Laparoscopic Cholecystectomy. JAMA Surg. 2023 Dec 01;158(12):1303-1310.
20. Maegawa FB, Stetler J, Patel D, Patel S, Serrot FJ, Lin E, Patel AD. Robotic compared with laparoscopic cholecystectomy: A National Surgical Quality Improvement Program comparative analysis. Surgery. 2025 Feb;178:108772.
21. Thangavelu A, Rosenbaum S, Thangavelu D. Timing of Cholecystectomy in Acute Cholecystitis. J Emerg Med. 2018 Jun;54(6):892-897.
22. Ke CW, Wu SD. Comparison of Emergency Cholecystectomy with Delayed Cholecystectomy After Percutaneous Transhepatic Gallbladder Drainage in Patients with Moderate Acute Cholecystitis. J Laparoendosc Adv Surg Tech A. 2018 Jun;28(6):705-712.

23. Woodward SG, Rios-Diaz AJ, Zheng R, McPartland C, Tholey R, Tatarian T, Palazzo F. Finding the Most Favorable Timing for Cholecystectomy after Percutaneous Cholecystostomy Tube Placement: An Analysis of Institutional and National Data. J Am Coll Surg. 2021 Jan;232(1):55-64.
24. Sperry C, Malik A, Reiland A, Thornburg B, Keswani R, Ebrahim Patel MS, Aadam A, Yang A, Teitelbaum E, Salem R, Riaz A. Percutaneous Cystic Duct Interventions and Drain Internalization for Calculous Cholecystitis in Patients Ineligible for Surgery. J Vasc Interv Radiol. 2023 Apr;34(4):669-676.
25. Storm AC, Vargas EJ, Chin JY, Chandrasekhara V, Abu Dayyeh BK, Levy MJ, Martin JA, Topazian MD, Andrews JC, Schiller HJ, Kamath PS, Petersen BT. Transpapillary gallbladder stent placement for long-term therapy of acute cholecystitis. Gastrointest Endosc. 2021 Oct;94(4):742-748.e1.
26. Troncone E, Amendola R, Moscardelli A, De Cristofaro E, De Vico P, Paoluzi OA, Monteleone G, Perez-Miranda M, Del Vecchio Blanco G. Endoscopic Gallbladder Drainage: A Comprehensive Review on Indications, Techniques, and Future Perspectives. Medicina (Kaunas). 2024 Apr 14;60(4)
27. Indar AA, Beckingham IJ. Acute cholecystitis. BMJ. 2002 Sep 21;325(7365):639-43