REVIEW ARTICLE

Classification and Management of Acute Cholangitis

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Abstract

Acute cholangitis (AC) is an urgent medical condition characterized by inflammation of the biliary system due to ascending bacterial infection that carries a high mortality rate. Obstruction precedes infection and is caused by both nonmalignant and malignant etiologies, but most are attributed to biliary stone obstruction or choledocholithiasis. AC is a clinical diagnosis based on patient presentation, laboratory values, and imaging criteria and is further stratified by severity. Treatment involves fluid resuscitation, antibiotics, and biliary drainage following the severity of the presentation. This review article describes the latest updates in the diagnosis, classification, and management of AC, as well as the outcomes of different treatment modalities.

Keywords: Acute cholangitis, Ascending cholangitis, Biliary drainage.

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Introduction

Acute cholangitis (AC), also known as ascending cholangitis, is an urgent medical illness brought on by a bacterial infection of the biliary system, with the common bile duct (CBD) obstruction being the primary cause of the majority of cases.[1] Multiple mechanisms lead to partial or complete biliary obstruction, each with different underlying risk factors contributing to AC morbidity and mortality.[2] Choledocholithiasis, which causes obstruction of the biliary tree, is the most significant risk factor for AC, representing almost 50% of cases.[3] Malignant obstruction, which accounts for 10–30% of cases of AC, is the second most frequent cause of biliary obstruction.[4] AC can also develop after intervention *via* endoscopic retrograde cholangiopancreatography (ERCP) for different biliary diseases, with a 0.5–2.4% postprocedure incidence rate.[5,6] Biliary stenting following ERCP also leads to stent-associated cholangitis (SAC) at a rate of 3.5–48.8% due to stent occlusion.[7] The risk of SAC also increased with length, size, and the number of stents placed.[8] Anatomic variations in patients, including multiple and hilar strictures, increase the risk of SAC and are associated with earlier onset of cholangitis. In patients with malignant biliary obstruction, the risk of SAC was 15.6%.[9] The overall current mortality rate of AC is estimated at 5–7.2% following the addition of biliary decompression *via* ERCP to therapy along with fluid resuscitation and antibiotics.[10]

Classic clinical signs of AC include Charcot's triad of fever, jaundice, and right upper quadrant abdominal pain; in late presentation, patients may sustain the Reynolds' pentad that adds to Charcot's triad signs of shock (hypotension and tachycardia) and an altered mental status.[2] However, Charcot's triad is said to have a specificity of over 90% and a sensitivity of about 36%, limiting its use as a diagnostic tool for AC.[11] The gold standard for biliary drainage in AC is biliary decompression *via* ERCP, but alternative percutaneous and surgical methods exist.[12]

Materials and Methods

The literature search was conducted in the PubMed, Scopus, Web of Science, and EBSCO electronic databases using combinations of the following keywords: AC, ascending cholangitis, optimized the search using specific conditions (and, in, and or). In an effort to highlight the most recent research, a date filter was applied to include papers after 2007 up until August 2022. Additional relevant papers were identified through reference lists of those articles and citation searching in Google Scholar for in-depth evaluation. The total number of articles was 982. Following primary exclusion criteria such as languages other than English, lack of access to a paper, or irrelevance to the subject of AC, case reports, commentaries, abstracts, and duplicate removal, we included a total of 172 papers composed of peer-reviewed articles related to the pathophysiology of AC, classification, and management in randomized controlled trials, case-control studies, cohort studies evaluating management guidelines, investigations, novel laboratory markers, and methods of management. After the final analysis, we selected 60 papers.

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Pathophysiology and Etiology

Acute cholangitis (AC) develops when an obstruction that prevents bile flow through the biliary tree occurs in conjunction with a superimposed bacterial infection due to ascending bacterial migration.[13] Bile itself, however, is considered sterile, as the concentration of bacteria present is insignificant and thus does not lead to an increase in the intraductal pressure.[13] Additionally, bile flow normally flushes microorganisms *via* an unblocked duct into the small intestine, preventing possible bacterial seeding.[13] Similarly, the sphincter of Oddi stops microorganisms from entering the CBD from the small intestine.[13] Bile salts also play a role in maintaining the sterility of bile by reducing bacterial reproduction.[13] Mechanistic barriers include the secretion of immunoglobulin A (IgA) and mucous from the biliary tree epithelium, which possibly serves as an anti-adhesive covering, as well.[13] Additionally, tight junctions between cholangiocytes and Kupffer cells on ciliary epithelium prevent bacteria migration from the hepatobiliary system to enter the portal venous system.[13] Obstruction reduces these defenses because the resulting increased intraductal pressure brought on by bile stasis expands tight junctions between cholangiocytes, and damages Kupffer cells, leading to decreased IgA synthesis locally. Bacterial seeding of bile then occurs due to the resulting biliary-venous and lymphatic reflux, allowing passage of bacteria into hepatic circulation culminating in bacteremia, endotoxemia, and eventually systemic inflammatory reaction.[13]

Diagnosis

The diagnosis of AC is made based on clinical presentation, laboratory investigations, and imaging studies.[13] The Tokyo Guidelines (TG) were the first to establish guidelines to diagnose and classify AC in 2007 and are regarded as the most reliable diagnostic standard. The TG 2018 (TG18) uses similar definitions as those in The TG 2013 (TG13) and currently requires evidence of systemic inflammation (fever or laboratory indicators), cholestasis, and imaging investigations demonstrating biliary dilatation or proof of a cause before considering a conclusive AC diagnosis (Tables 1 and 2).[14] The diagnostic performance of TG18 in identifying definite cholangitis recently showed an 82% sensitivity but had low specificity at 60%.[15]

Investigations

An elevated white blood cell (WBC) count and predominantly cholestatic liver pattern abnormalities are two laboratory indicators used to diagnose AC.[16] TG18 suggested procalcitonin (PCT) as a prognostic marker to determine the severity of AC and the necessity for urgent biliary decompression.[14] There is insufficient evidence to support the clinical utility of PCT in determining the severity of AC, even though higher PCT levels were consistently reported in severe AC compared to mild and moderate AC in numerous studies.[17] However, the evidence to determine appropriate serum cutoff points for urgent intervention is inconsistent.[18]

A recently published study proposed the use of the neutrophil-lymphocyte ratio (NLR) as a potential early predictive marker for AC. In contrast to the total WBC count, NLR was recently shown to be more accurate in reflecting immune and inflammatory responses. An elevated NLR correlated with severe AC, shock, and positive blood culture, suggesting that serial NLR measurements may be used to monitor AC progression in severe cases and possibly predict the occurrence of shock. The applicability of the results may be limited in patients with concurrent conditions with basal or superimposing elevations in inflammatory markers and necessitates further exploration through multicenter studies and in larger patient populations.[19]

Presepsin has recently been identified as a potential biomarker of bacterial infectious illness. Studies have shown that it has higher sensitivity and accuracy than PCT, WBC count, and C-reactive

Table 2: Severity assessment criteria for AC adapted from TG18/TG13

Grade	*Clinical features*	*Labs*
I (mild)	Does not meet the criteria of "grade III (severe)" or "grade II (moderate)" AC at initial diagnosis	–
II (moderate)	AC + any 2 of the following conditions:	1. Abnormal WBC count (>12000/mm^3 or <4000/mm^3) 2. Fever (≥39°C) 3. Age (≥75 years) 4. Hyperbilirubinemia (total bilirubin ≥5 mg/dL) 5. Hypoalbuminemia (<STD × 0.7)
III (severe)	AC + one dysfunction at least in any of the following systems:	1. Cardiovascular—hypotension requiring vasopressors (dopamine >5 ug/kg per min or any dose of norepinephrine) 2. Neurological—disturbance of consciousness 3. Respiratory PaO_2/FiO_2 <300 (arterial oxygen partial pressure to fractional inspired oxygen ratio) 4. Renal—oliguria, serum creatinine >2.0 mg/dL 5. Hepatic—prothrombin time-international normalized ratio >1.5 (prothrombin time-international normalized ratio). 6. Hematologic—platelet count <100000/mm^3

STD, standard deviation

Table 1: Diagnostic criteria for AC adapted from TG18/TG13[14]

A. Systemic inflammation	*B. Cholestasis*	*C. Imaging*
A-1. Fever (temperature >38°C) and/or shaking chills A-2. Evidence of inflammatory response. WBC count <4000 or >10000/mm^3, C-reactive protein >/10mg/L	B-1. Jaundice. Total bilirubin ≥20 mg/L B-2. Abnormal liver function tests. Aspartate aminotransferase, alanine transaminase, alkaline phosphatase, gamma-glutamyl transpeptidase (>1.5 × STD)	C-1. Biliary dilatation C-2. Evidence of etiology on imaging (stricture, stone, stent, and others)

Suspected diagnosis: one item in A + one item in either B or C; definite diagnosis: one item in A, one item in B + one item in C protein (CRP).[20] Presepsin had a higher sensitivity and specificity for predicting bacterial blood cultures and better sensitivity and specificity for stratifying AC. Preseptsin performed better than CRP and PCT as a biomarker of infection, predicting infection disease and risk stratification. In a single-center AC patient population, presepsin levels on admission also closely correlated with TG18 scores, suggesting that presepsin levels in the emergency department (ED) during AC were related to organ dysfunction, suggesting a role for the biomarker in AC risk stratification in early and acute settings.[20] However, its clinical utility and ability to predict patient mortality early require more extensive studies. Levels of another bacterial infection biomarker, lipocalin-2 (LCN2), recorded once at admission, were recently investigated in AC patients at admission to explore its predictive value and potential ability to facilitate earlier biliary drainage through risk stratification in the ED. The preliminary study showed LCN2 to be a more significant risk factor for severe AC when compared to the predictive values of other inflammatory markers, including WBC, CRP, PCT, and NLR. Future studies investigating serial LCN2 measurements, as well as the use of larger patient samples, will be required to more thoroughly explore and validate the use of LCN2 in the early assessment and management of severe AC.[21]

Imaging

In patients with jaundice, ultrasonography is commonly used and considered the best imaging tool.[22] Both the sensitivity and specificity of ultrasound exceed 95% for the detection of gallstones.[23] However, when the CBD is dilated, ultrasonography is 75% sensitive for choledocholithiasis and 50% sensitive when the CBD is of normal caliber. It drops to 40% for smaller stones or stones lodged close to the ampulla when intestinal gas is present.[24] CT may be more useful in ruling out mechanical obstruction after recent surgery, trauma, or biliary intervention.[25] To localize and characterize duct pathology, magnetic resonance cholangiopancreatography (MRCP) is ideal.[26]

The use of MRCP allows for imaging of the biliary system in a noninvasive and safe manner that does not require the use of anesthesia or radiation and allows for better visualization of ducts proximal to the obstruction.[27] The technique utilizes T2-weighted images resulting in a substantial contrast between stationary fluids, in this case, bile, and the background. As a result, a strong signal is presented by the bile in comparison to a low signal intensity emitted by the background. Moreover, blood doesn't affect the result since no signal comes from it.[28]

The technique is also useful for evaluating biliary enteric anastomosis, which is usually difficult because of modified anatomy beyond the duodenum level. MRCP can display the location of the anastomosis and stones, as well as the status of the intrahepatic ducts.[27] The periductal transient attenuation difference was an independent predictor of AC. MCRP can be used to detect significant differences in wall thickness. However, it has limited utility in detecting duct dilation compared to other imaging modalities.[29]

Alternatively, hepatobiliary iminodiacetic acid (HIDA) scans can be implemented, which use radiotracers to image the biliary system. The utility of the scan is that it allows for the evaluation of biliary pathologies, such as obstructions, by following radiotracers as they move through the bilirubin metabolic pathway and into the bile ducts.[30] HIDA is used to evaluate for cholecystitis, biliary obstruction, and bile leaks. The main limitation is its lack of anatomical detail. Hence, it is commonly used as an additional study with other described modalities, limiting its utility in AC.

Management

Antimicrobial Therapy

The current recommendation for antimicrobial therapy duration after source control in patients with AC is four to seven days, according to TG18, albeit there is little evidence to support this. Several reports have since emerged supporting the use of short-term administration of antimicrobial therapy with satisfactory results. The national sepsis guidelines from the Netherlands, however, recommend only a three-day antibiotic course or less in patients who successfully underwent ERCP.[31] These recommendations mirror a recent retrospective study by Satake et al. that demonstrated the efficacy of antibiotic therapy for three days or less after biliary drainage.[32] More recently, a two-day course of antimicrobial therapy after successful ERCP was shown to be just as effective as a four-day course in patients with milder and moderate cases of AC.[33]

The TG18 also recommends a 2-week course of antibiotic therapy for AC cases with gram-positive cocci (GPC)—positive blood cultures due to the possibility of developing infective endocarditis (IE), but this risk was not found to be significant in a study by Gomi et al., in which no IE was observed in 243 cases of GPC-positive blood culture treated with a short course of antibiotic therapy.[34] Thus, even patients with GPC-positive blood cultures may need only a shorter course of antibiotics to treat cholangitis.[34]

Empiric AC therapy recommendation from the TG18 includes third-generation cephalosporines, piperacillin/tazobactam, or carbapenems as first-line agents, taking into consideration local susceptibility data and infection severity.[34] Here, bile cultures collected during ERCP can assist guide antibiotic therapy and decrease the duration of treatment.[31,35] Antibiotic coverage after empiric therapy largely depends on the source of infection and is further stratified into community-acquired versus healthcare-associated infections.[36] Patients with community-acquired cholangitis with milder presentations maybe be sufficiently covered under piperacillin/tazobactam.[36] Coverage for penicillin-resistant gram-negative bacteria may be necessary for patients with more complicated cases, such as those with high comorbidity load.[36]

Antifungal therapy may also be added to the treatment regimen in patients with healthcare-associated infections and biliary stents.[34]

While serum PCT and CRP are significant markers postoperatively to consider discontinuing antibiotics, there is no impact on the outcome of AC.[37] Soluble triggering receptor expressed on myeloid cells 1 (sTREM-1) is the best biomarker to monitor patients' response to antimicrobial therapy and biliary drainage.[38]

Biliary Drainage

Endoscopic Biliary Drainage (EBD)

Persistent obstruction of the biliary system could result in portal hypertension, bile reflux, bacterial bloodstream infection, and AC.[16] Therefore, the blockage must be relieved with biliary decompression and drainage to stop additional systemic organ damage and improve treatment outcomes.[39] AC can be controlled at the source with prompt biliary system draining, which lowers bile and serum endotoxin levels and increases biliary excretion of IgA and antibiotics.[40] Early biliary draining improves clinical outcomes and decreases 30-day mortality, hospital and intensive care unit stays, organ dysfunction, and fever duration; these effects were independent of susceptibility to empiric antibiotic use.[40] Based on TG18, the American and European Society of Gastrointestinal Endoscopy (ASGE and ESGE) guidelines on CBD stones currently recommend performing an ERCP to obtain biliary drainage in case of AC.[41]

Endoscopic transpapillary biliary drainage (EBD) under ERCP is still regarded as the gold standard for biliary decompression. Other EBD modalities include endoscopic sphincterotomy (EST), endoscopic biliary stenting (EBS), and endoscopic nasobiliary drainage (ENBD) for tube insertion prior to or concurrently with ERCP to prevent post-ERCP pancreatitis.[42] EBD without EST, however, is generally necessary for the event AC is combined with disseminated intravascular coagulation (DIC) due to the high risk of post-EST bleeding.[43] ENBD involves using external drainage, which relies on catheters to monitor, aspirate, and washes bile and may be beneficial to patients with a high APACHE II score or a high total bilirubin level. In elderly or confused patients, however, ENBD has the disadvantage of self-extraction of the transnasal catheter due to discomfort.[43] Alternatively, emergent radiation-free EBD is an effective and safe option for critically ill patients with severe cholangitis who are hemodynamically unstable or on mechanical ventilators and cannot be transported out of intensive care units and was more recently shown to be as effective as percutaneous drainage.[44] Almost 90% of AC cases can be successfully treated with ERCP, with a 5% complication rate and a 1% mortality rate.[45] Relative contraindications include cardiopulmonary instability, coagulopathy, pregnancy, and severe contrast allergy.[46] When coagulopathy cannot be addressed before the procedure, sphincterotomy is not recommended.[47]

Although most patients with biliary tract obstruction who develop mild to moderate cholangitis respond well to antibiotics, 15–30% of patients develop severe cholangitis and require urgent drainage.[48] It is critical to treat patients with acute biliary tract infections according to their severity since treatment timing greatly influences subsequent progression.[40]

For moderate and severe cholangitis, TG18 recommends urgent or emergent biliary drainage; for mild AC, it is only advised if antibiotics are ineffective.[49] Although the ideal time for biliary drainage is not established, most experts concur that biliary decompression should be done within 48 hours.[49] A retrospective study on the best time to have an ERCP revealed that, compared to delayed drainage (after 24 hours), early draining within the first 24 hours resulted in a lower incidence of organ failure, a shorter hospital stay, shorter antibiotic therapy, and a lower mortality rate.[50] According to more recent evidence, a meta-analysis showed that emergent ERCP conducted within 12 hours improves death rates, which aligns with the ESGE standards that advise ERCP within 12 hours for septic shock patients.[41,51] A retrospective analysis with a large sample size also demonstrated that drainage within 48 hours was related to improved in-hospital mortality (IHM).[52] However, a retrospective study of 6063 AC patients conducted by Kiriyama et al. revealed decompression within 24 or 48 hours improved the 30-day mortality rate only in grade II patients.[53]

It is difficult to predict which individuals would develop sepsis and die from grade III AC or severe cholangitis despite receiving medical treatment and which ones will require decompression.[6] Until recently, there was no evidence to support the benefits of grade III AC patients having biliary drainage early (within 12 hours).[53] Lu et al. demonstrated that early biliary drainage (within 24 hours) was advantageous for AC patients with grade III severity and further categorized the severity of AC in grade III patients depending on organ dysfunction levels to establish the most optimal timing for endoscopic intervention. In instances worsened by cerebral and cardiovascular dysfunction, as well as in patients with lactate >2 mmol/L, aberrant WBC count, hyperbilirubinemia, or hypoalbuminemia, early drainage within 12 h of arrival has been found to lower hospital mortality regardless of the severity of AC.[54]

Percutaneous Transhepatic Biliary Drainage (PTBD)

Percutaneous drainage is regarded as a second-line therapeutic option in situations when ERCP has failed, in patients with several comorbidities, or in those whose altered, or variant anatomy makes endoscopic therapy challenging. Unlike ERCP, PTBD can target specific ducts to maximize the drainage of functional liver parenchyma, which is seen when the obstruction is above the CBD.[42] Due to the lack of anesthetic or intravenous sedation, clinically unstable patients may be candidates for this procedure.[42] Percutaneous drainage has several drawbacks, such as pain for the patient, drain dislocations and occlusion requiring reintervention, and prolonged hospital stays. Long-term drain placement also leads to poorer quality of life and a higher risk of complications like sepsis, biliary peritonitis, and intraperitoneal hemorrhage.[55] Contraindications to PTBD include ascites, intrahepatic biliary obstructions, and coagulopathies.[13]

EUS-guided Biliary Drainage (EUS-BD)

In cases where ERCP has failed, or PTBD is contraindicated, a novel approach that recently gained traction is EUS-BD.

The procedure can be carried out in a variety of ways, mostly *via* an intra- or extrahepatic route. The left lobe of the liver is reached through the stomach in the intrahepatic approach, and either a transpapillary stent is implanted using the EUS-rendezvous technique (EUS-RV) or a transluminal stent is implanted using a hepaticogastrostomy.[56] The choledochoduodenostomy with a transluminal stent or the rendezvous procedure with a transpapillary stent can both be performed using the extrahepatic route, which gains access to the bile duct through the duodenal wall. The rendezvous stenting technique has the benefit that the entire process can be carried out endoscopically, without the need for a scope, through a temporary fistula made between the upper intestine and the intrahepatic bile ducts.[56]

Recent studies have shown its advantage over PTBD, resulting in significantly lower acute and total adverse events, including bleeding, sepsis, and fistula formation. Furthermore, it is a less invasive procedure that does not require the installment of a long-term drain which reduces the need for re-intervention. However, its technicality and complexity, as well as the specialized training required to perform the procedure, limits its scalability, but this has not hampered its utilization as the preferred modality in the event of ERCP failure due to its lower risk profile in comparison to PTBD.[55]

Surgery

Traditional surgical techniques are intrusive, time-consuming, and may harm a patient's capacity to recover, especially in old and fragile patients who may have a reduced tolerance for surgery owing to diminished physiological function and may consequently be at risk for consequences. Thanks to improvements in endoscopic technology and minimally invasive procedures, less trauma, quicker recovery, and better treatment outcomes are all advantages of less invasive surgical techniques.[47]

Open surgical drainage is rarely done but is employed when ERCP, EUS, or PTBD are contraindicated or if these procedures are unsuccessful.[42] Since laparoscopic surgery has minimal incisions and a high prognosis, it was introduced as an intervention to treat bile tract illnesses. Compared to laparotomy, laparoscopic surgery involves fewer surgical procedures and takes less time to complete. Compared to traditional surgery, laparoscopic surgery was linked to a significant improvement in treatment effectiveness, a decreased risk of complications, and better treatment results in patients with severe AC.[57]

For the treatment of patients with CBD stones in elective conditions, laparoscopic CBD exploration (LCBDE) is a well-established surgical method which can drain the bile duct and remove CBD stones.[58] The standard four-trocar operative is used for LCBDE. After the Calot's triangle is explored and presented, a longitudinal incision along the CBD is made. The CBD is irrigated to remove the stones. Second, the choledochoscope removes the stones and allows them to explore the remnant ones. If the remnant stones cannot be removed, they are removed postoperatively.

In emergent situations such as AC, early LCBDE within 72 hours of diagnosis may be superior to delayed procedure due to the shorter LOS and less cost.[58] Improvement of symptoms was achieved in all patients with LCBDE. None of the patients developed stenosis of the CBD after LCBDE.[58]

Laparoscopic surgery had a greater effective rate, a reduced incidence of complications, and improved symptom alleviation and postoperative inflammation for patients with AC.[59] Endoscopic surgery was considerably superior to laparotomy in terms of the duration of abdominal pain relief, time spent getting out of bed, frequency of complications, and length of hospital stay.[60] In addition, laparoscopic surgery significantly improved WBC, total bilirubin, and alanine transaminase levels in AC patients compared to standard laparotomy. Additionally, laparoscopic surgery was superior to laparotomy for postoperative inflammatory recovery in acute and severe conditions, suggesting that the former may lessen the risk of toxins entering the blood circulation and the spread of inflammation.[57]

Conclusion

Acute cholangitis (AC) represents an urgent medical illness where CBD obstruction is present, usually due to choledocholithiasis (50% of cases) or malignant obstructions (10–30%). Its diagnosis is based on clinical presentation, laboratory investigations, and imaging studies. The Tokyo Guidelines classification system is used in most services to stratify the severity and establish the best treatment option for each case. The treatment's pillars are antibiotics associated with biliary tract decompression, usually performed by ERCP. Depending on availability, other treatment modalities can be used, like the percutaneous approach and surgery. The mortality rate ranges from 5–7.2%, with a higher mortality rate in patients who require emergency biliary decompression or surgery.

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