Study on acalculous cholecystitis in critically ill patients.

Abstract

Introduction: Acute acalculous cholecystitis (AAC) may be a serious complication of critical illness. We evaluated the underlying diseases, clinical and diagnostic features, the severity of associated organ failures, and the outcome of operatively treated AAC during a mixed ICU patient population.

Aim: To study acalculous cholecystitis in critically ill patients with given data.

Method: The data of all ICU patients who had operatively confirmed AAC during their ICU stay between 1 January 2021 and 31 July 2021 were collected from the hospital records and the intensive care unit's data management system for further analysis.

Results: Two hundred and eighteen cases of acute cholecystitis with complete charts were available for analysis. The study group included 67 critically ill patients with ASA 3 and 4, while the control group included 150 fit patients with ASA 1 and 2. Both groups were comparable with regard to preoperative data. Histopathology confirmed severe cholecystitis in a significant number of cases in the study group compared to the control group. Significantly higher rates of morbidity and mortality were recorded in the study group. Equally, significantly more patients from the study group were managed in the ICU.

Conclusion: Acute acalculous cholecystitis was associated with severe illness, infection, long ICU stay, and multiple organ failure. Mortality was related to the degree of organ failure. Prompt diagnosis and active treatment of AAC are often life-saving in these patients.

Keywords: acute cholecystitis, gallbladder diseases, acalculous cholecystitis, surgical intensive care, patient care, critical care.

Introduction:

Acute cholecystitis may be a pathology during which the gallbladder wall becomes inflamed. Gallstones are the first triggering think about 90% of the causes of cholecystitis, they're present in

additional than 10% of the population and their presence increases with age. the most factors for the formation of gallstones are DM, estrogen, pregnancy, cirrhosis, obesity, and hemolytic disease. However, in approximately 2 to fifteen cases, acute cholecystitis can occur without the presence of gallstones, and these are named acute acalculous cholecystitis (AAC), a condition that's diagnosed with increasing frequency in critical patients and is reported worldwide

AAC is predominant in male patients aged 50 years old and over (80% of cases). it's reported within the postoperative period of non-traumatic surgery, in burn patients, and in association with sepsis, DM, vasculitis (including polyartheritis nodosa), and prolonged use of parenteral nutrition. Untreated, its short-term mortality can reach 35%, while long-term mortality rate can reach 59%. There are reports of its emergence in previously healthy children and in children with infection or trauma, AAC corresponds to 30 to 70% of acute cholecystitis.

The incidence of AAC following open abdominal aortic reconstruction varies between 0.7% and 0.9%; moreover, this condition has been reported as a complication in endovascular aortic repair. Approximately eighty percent (80%) of patients diagnosed with AAC following surgery within the absence of trauma are men.

AAC usually occurs after severe clinical disease or trauma, like extensive burns, polytrauma, operation, end-stage renal disease (ESRD), leukemia, and severe infection. However, the event of AAC isn't limited to surgical patients, traumatic wounds, or critical illness. Patients with diabetes[1]thanks to cholesterol and post-hemorrhagic shock resuscitation are diagnosed with AAC[4]. Besides these conditions, ESRD may be a predisposing factor, because DM and atherosclerosis are common within the final of the disease, which frequently evolves with low-flow hemodialysis. Hemorrhagic AAC has been reported in patients with ESRD, especially when associated with any uremic thrombocytopenia or frequent exposure to uremic heparinoids. Research indicates that the event of a secondary infection of the gallbladder: can involve disseminated candidiasis or leptospirosis; and occurs during systemic sepsis, in patients with chronic biliary tract diseases, such as typhoid and non-typhoid Salmonella typhoid; and through diarrhoeal diseases, like cholera or Campylobacter enteritis and tuberculosis. Cases of AAC have also been reported with malaria, brucellosis, Coxiella burnetii, and dengue. Several viral pathogens are related to AAC, including hepatitis A, hepatitis B, and Epstein-Barr virus[2]

Method:

This work consists of a literature review in the databases PubMed, Scielo, Scopus, and Web of Science. Data was collected on case reports, cohort studies, and literary reviews, using the

keywords “acute cholecystitis, gallbladder diseases, acalculous cholecystitis, surgical intensive care, patient care, critical care”.

The data of all ICU patients who had operatively confirmed AAC during their ICU stay between 1 January 2021 and 31 July 2021 were collected from the hospital records and the intensive care unit's data management system for further analysis.

A detailed analysis of both the databases, literature and data of the patient was taken and a conclusion was arrived at [3]

Result:

Half of the patients with AAC may present additional complications at the time of diagnosis, such as gangrene, gallbladder perforation, empyema, and emphysematous cholecystitis. These conditions are more common with AAC than acute calculous cholecystitis, and should always prompt suspicion of AAC. When undiagnosed, clinical deterioration is usually rapid and catastrophic.

Despite the fact that many diagnosed patients are not in good enough health for surgery, a temporary percutaneous cholecystostomy is frequently performed. It is an invasive surgery that involves the decompression of the gallbladder and the management of inflammation. The cholecystostomy tube is left in place until the patient is clinically healthy enough to undergo a cholecystectomy, which usually takes 6-8 weeks following acute cholecystitis recovery.

This surgery has a low mortality rate of less than 3%. Percutaneous drainage can completely decompress the inflamed gallbladder, resulting in a quick relief in symptoms. According to recent studies, more than 90% of patients have symptom improvement following three days of draining.

The most common complications with this procedure are biliary tract rupture, pain at the puncture site, catheter hemorrhage or dislocation, and recurrence of the condition after removal of the percutaneous drainage catheter in patients with AAC, which occurs in 21.7 to 46.7 percent of patients over a 12- to 37-month follow-up period.

Discussion:

Etiology: The most commonly isolated microorganisms are E. coli and other gram-negative enteric bacteria, but dengue viruses, cytomegalovirus, varicella-zoster, Epstein-Barr virus, hepatitis A virus, Leptospira sp., Salmonella sp., Vibrio cholera, Coxiella Burnetti, Plasmodium falciparum, Cryptosporidium, and Candida sp. have all been linked

People with HIV who have opportunistic infections including CMV, Cryptosporidium, Mycobacterium tuberculosis, Mycobacterium avium intracellulare, or fungal infections are at an increased risk of acquiring AAC.

There have been reports of uncommon cases caused by Lactococcus garvieae, a pathogenic agent in fish, as well as a rare complication in Salmonella infection cases, with a maximum incidence of 0.6 percent isolation. The development of the latter pathology is based on gallbladder ischemia and cystic artery occlusion.

Pathophysiology: AAC's etiology isn't well understood, and its specific mechanism is unknown. Several causes, including ischemia, infection, and biliary alterations, have been implicated in studies. As a result, AAC has a complex etiology.

Due to poor mucosal resistance, patients with visceral atherosclerosis may be at a greater risk of AAC. AAC can also arise in people with sepsis or who are immunocompromised. Diabetes mellitus is another well-known risk factor. Trauma, burns, major surgery, particularly involving the cardiac valves, extended parenteral feeding, vasculitis (including polyarteritis nodosa), opiate usage, and positive pressure ventilation have all been linked to the development of the condition.

The cause of the disease is unknown, although one theory is that the bile of individuals with severe cholestasis, who are usually not fed enterally, causes distention and increased tension inside the gallbladder wall, affecting blood flow. The presence of bacterial proliferation and the activation of pro-inflammatory mediators, in addition to the frequent occurrence of ischemia, might result in gallbladder gangrene. Another possibility, as documented by Mourani et al. in their instance of hepatitis A-related cholecystitis, is direct viral invasion of the gallbladder.[5]

Given these findings, it's crucial to call attention to the four factors that appear to be most strongly linked to the start of AAC: 1) biliary stasis - biliary stasis is caused by changes in bile composition and gallbladder damage caused by alterations in the smooth muscle of the gallbladder and sphincter of Oddi dysfunction. 2) systemic inflammatory response – the production of inflammatory mediators such as factor XII and platelet-activating factor in response to systemic damage causes an inflammatory process in the gall bladder. 3) bile acid production and secretion are reduced due to visceral ischemia-reperfusion damage and 4) infectious diseases – tuberculosis should be considered in any differential diagnosis, especially in immunocompromised patients, while Salmonella is a likely infectious agent when diarrhea is present, as it can spread through the blood or lymph system even weeks after the initial infection. The remaining agents have already been mentioned. It's important noting that AAC can impact youngsters who have been infected with a virus.

The evolution of AAC following an acute Epstein-Barr virus (EBV) infection is poorly known. The main causes involved, however, are believed to be direct EBV infection and/or gallbladder

inflammation due to bile stasis. AAC appears to be more common in young women with an acute EBV infection. It's worth noting that the presence of upper respiratory symptoms and hematological data consistent with viral infection can aid in the diagnosis of EBV-related AAC. The presence of clinical data, laboratory findings, and ultrasound findings that are compatible with AAC, on the other hand, can lead to diagnostic conundrums and wasteful therapy processes.

Clinical status: The commencement of the illness is marked by pain in the right hypochondrium that radiates to the scapula and epigastric region. In up to 70% of patients, fever, nausea, and vomiting are present.[6]

In the context of high fever, chills, leukocytosis, and reduced peristalsis, the formation of abscesses, poor perfusion, or gangrene in the gallbladder should raise suspicions. Because many of these symptoms are common in patients with serious illnesses who require sedation, the clinical examination can be hampered, preventing timely identification.

A palpable lump, indicating an enlarged gallbladder, can be found in up to 20% of patients. Murphy's sign may also be present, indicating peritoneal visceral-parietal compromise.

Other symptoms may include fever, nausea, vomiting, diarrhea, dyspepsia, exhaustion, altered mental status, and jaundice, however, they are highly nonspecific.

Diagnosis: Because AAC is a rare condition, it is frequently misdiagnosed, which has a significant impact on its mortality (10-50%) as compared to acute calculous cholecystitis (about 1%).

Except for the absence of gallstones, the clinical and radiological signs of AAC are the same as those of acute calculous cholecystitis. When patients appear with stomach discomfort, fever, and unexplained leukocytosis and sepsis in critically hospitalized or chronically ill patients, the diagnosis should always be evaluated.[7]

Because it is difficult to diagnose, the level of suspicion is usually high, and it is usually verified after a clinical examination, laboratory results, and radiographic and/or surgical findings [55, 63].

Other tests show elevations in transaminases, alkaline phosphatase, bilirubin, and amylase, while the hemogram may show leukocytosis with the left shift.

There is now a significant debate about the best image mode and the initial mode to utilize when diagnosing AAC. It's worth noting that no one radiological mode is sensitive or specific enough to validate a diagnosis.

Because of its great sensitivity for gallstones and aberrant wall thickness (> 3 mm), ultrasound is the imaging exam of choice when starting an inquiry. Murphy's sign is also visible on ultrasonography, which shows a tight and swollen gallbladder, as well as pericholecystic fluid and the lack of gallstones. The sensitivity and specificity of this test might range from 30% to 100%.

Additionally, an ultrasound can detect biliary sludge, gallbladder distention greater than 0.5 cm, gallbladder striation, mucosal peeling, emphysematous cholecystitis, and gallbladder perforation. With this CT scan, abdominal radiology is valuable[8].

Treatment: It is important to note that, despite the implementation of quick and sufficient treatment, the death rate for patients with AAC is around 30% due to their serious clinical status and rapid progression to gangrene and perforation.

Because the condition has a high risk of catastrophic progression, an emergency cholecystectomy, either open or laparoscopic, is the chosen treatment for AAC. In the absence of comorbidities, careful antibiotic therapy with Salmonella enteritis infection is the best therapeutic approach in situations where the etiology of AAC is Salmonella enteritis infection. The symptoms are normally resolved after a 4- to 6-week course of broad-spectrum antibiotics. Cholecystectomy is not necessary if the symptoms go away and the control ultrasound shows a non-dilated gallbladder with a thin wall. Patients who are given a long course of broad-spectrum antibiotics usually have a positive outcome.

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Dengue patients should have conservative treatment because they do not have diffuse peritonitis or perforation at the time of diagnosis. Patients who have a cholecystectomy or a percutaneous cholecystostomy may experience bleeding that necessitates a transfusion and even demands a longer stay in the hospital.

If the patient is a surgical candidate, a laparoscopic cholecystectomy should be performed as soon as possible, since this minimizes inpatient time and complication rates while leaving mortality unaffected. When there is no association between the symptoms and the ultrasound findings, cholecystectomy is preferable to cholecystostomy because of the risk of necrosis and gangrene. Hospitalization, fasting, hydration, and electrolyte replenishment are all suggested, as is analgesia for pain management [1]. Morphine and its derivatives, however, are not indicated due to the danger of sphincter of Oddi spasms. Antibiotics are indicated, with gram-negative and anaerobic microbes included; the well-documented participation of Escherichia coli is worth mentioning once more[9].

Conclusion:

The death rate of individuals with acute acalculous cholecystitis remains high, regardless of therapy, owing to serious underlying medical problems and the disease's rapid progression to gangrene and perforation.

Despite the fact that it is a rare and difficult pathology to diagnose, a good diagnosis can be made with a thorough initial assessment and additional tests.

Cholecystectomy with drainage of any accompanying abscesses is the definitive therapeutic approach, while cholecystostomy is typically chosen due to its less invasive nature.

The lack of published works on this pathology limits our understanding of acute acalculous cholecystitis, indicating that more research is needed to improve the current understanding of its pathophysiology, risk factors, and treatment management, resulting in a better understanding of the disease's many aspects.

Ethical considerations:

Before starting the study, the Institution Review Board of Saveetha University has approved our protocol, later grant sanction form was obtained from HOD's of all department. Further informed oral consent was obtained from all the patients before they were included in the study.

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