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Methylprednisolone as a Novel Adjuvant Therapy for Acute Cholecystitis with Non-obstructive Jaundice: A Case Report

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A B S T R A C T

Acute cholecystitis is a common inflammatory condition of the gallbladder, primarily caused by gallstones or infection. While jaundice is often associated with acute cholecystitis due to biliary obstruction, it can also occur without evidence of obstruction, posing a diagnostic and therapeutic challenge. This case report presents a patient with acute cholecystitis and non-obstructive jaundice who demonstrated a remarkable response to methylprednisolone therapy. A 25-year-old male presented with a three-week history of right upper quadrant abdominal pain, jaundice, nausea, gray-colored stools, and dark urine. Physical examination revealed icterus, right upper quadrant tenderness, and a positive Murphy's sign. Laboratory investigations showed elevated total and direct bilirubin levels, transaminitis, and an increased gamma-glutamyl transferase (GGT) level. Abdominal ultrasonography confirmed acute cholecystitis with sludge but no biliary obstruction. The patient was treated with intravenous fluids, antibiotics, and supportive care. On the fourth day of hospitalization, methylprednisolone (62.5 mg twice daily) was initiated due to persistent jaundice and transaminitis. The patient's clinical and laboratory parameters improved significantly following the initiation of methylprednisolone. In conclusion, this case suggests that methylprednisolone may be a valuable adjuvant therapy for acute cholecystitis with non-obstructive jaundice, particularly in cases with evidence of liver and bile duct inflammation. Further studies are needed to confirm these findings and establish the optimal dosage and duration of methylprednisolone therapy in this setting.

1. Introduction

Acute cholecystitis, a prevalent inflammatory condition affecting the gallbladder, is primarily caused by gallstones or infections. The typical clinical presentation of acute cholecystitis includes right upper quadrant pain, fever, nausea, and vomiting. Jaundice, characterized by yellow discoloration of the skin and sclera due to elevated bilirubin levels, is often associated with acute cholecystitis. In most cases, jaundice in acute cholecystitis results from common bile duct obstruction by gallstones, leading to impaired bile flow and bilirubin accumulation. However, jaundice can also occur in acute cholecystitis without evidence of biliary obstruction, a condition known as non-obstructive jaundice. The pathogenesis of non-obstructive jaundice in acute cholecystitis is complex and not fully understood. Proposed mechanisms include direct inflammation of the liver and bile ducts, systemic inflammation affecting bilirubin metabolism, and sepsis-induced cholestasis. The management of acute cholecystitis with non-obstructive jaundice can be challenging. While cholecystectomy remains the definitive treatment for acute cholecystitis, the role of adjuvant therapies, such as steroids, is less clear.

Steroids have potent anti-inflammatory effects and have been used in various liver diseases to reduce inflammation and improve liver function. However, their use in acute cholecystitis with non-obstructive jaundice has not been extensively studied.[1-3]

Acute cholecystitis is a common surgical emergency, with an estimated incidence of 10-20 per 100,000 population per year. The prevalence of acute cholecystitis varies depending on several factors, including age, gender, ethnicity, and dietary habits. The condition is more common in women, with a female-to-male ratio of 2:1. The risk of acute cholecystitis increases with age, with the highest incidence occurring in individuals over 60 years old. The pathogenesis of acute cholecystitis typically involves a combination of factors, including gallstone formation, gallbladder obstruction, and bacterial infection. Gallstones, composed primarily of cholesterol, bile pigments, and calcium salts, can obstruct the cystic duct, leading to gallbladder distension, inflammation, and infection. The inflammatory response in acute cholecystitis is mediated by various cytokines and chemokines, leading to the recruitment of neutrophils and other immune cells to the gallbladder wall. The clinical presentation of acute cholecystitis can vary depending on the severity of the inflammation and the presence of complications. The most common symptom is right upper quadrant pain, which may be constant or intermittent and may radiate to the right shoulder or back. Other symptoms include fever, nausea, vomiting, and anorexia. Physical examination may reveal right upper quadrant tenderness, a positive Murphy's sign, and guarding or rigidity.[4-6]

The diagnosis of acute cholecystitis is based on a combination of clinical findings, laboratory tests, and imaging studies. Laboratory tests may reveal leukocytosis, elevated liver function tests, and elevated bilirubin levels. Imaging studies, such as abdominal ultrasonography or computed tomography (CT) scan, can confirm the diagnosis of acute cholecystitis and rule out other conditions. The treatment of acute cholecystitis typically involves a combination of supportive care, antibiotics, and cholecystectomy. Supportive care includes intravenous fluids, pain management, and antiemetics. Antibiotics are used to treat bacterial infections and prevent complications. Cholecystectomy, the surgical removal of the gallbladder, is the definitive treatment for acute cholecystitis.[7-8]

Jaundice, a yellow discoloration of the skin and sclera due to elevated bilirubin levels, is often associated with acute cholecystitis. In most cases, jaundice in acute cholecystitis results from common bile duct obstruction by gallstones, leading to impaired bile flow and bilirubin accumulation. However, jaundice can also occur in acute cholecystitis without evidence of biliary obstruction, a condition known as non-obstructive jaundice. The pathogenesis of non-obstructive jaundice in acute cholecystitis is complex and not fully understood. Proposed mechanisms include direct inflammation of the liver and bile ducts, systemic inflammation affecting bilirubin metabolism, and sepsis-induced cholestasis. The management of acute cholecystitis with non-obstructive jaundice can be challenging. While cholecystectomy remains the definitive treatment for acute cholecystitis, the role of adjuvant therapies, such as steroids, is less clear. Steroids have potent anti-inflammatory effects and have been used in various liver diseases to reduce inflammation and improve liver function. However, their use in acute cholecystitis with non-obstructive jaundice has not been extensively studied.[9-10] This case report describes a patient with acute cholecystitis and non-obstructive jaundice who demonstrated significant clinical and laboratory improvement following treatment with methylprednisolone, a synthetic glucocorticoid with potent anti-inflammatory properties.

2. Case Presentation

A 25-year-old male presented to our hospital with a three-week history of right upper quadrant abdominal pain. The pain was described as constant, stabbing in nature, and had worsened over the past two days. The pain was aggravated by movement. The patient also reported jaundice of three weeks duration, characterized by yellowing of the skin and sclera. Accompanying the pain and jaundice were nausea and decreased appetite. Additionally, the patient reported passing gray-colored stools and dark, "tea-colored" urine. The patient denied any significant past medical history. He was not taking any medications and reported no known allergies. Social history revealed regular alcohol consumption. There was no family history of liver or gallbladder disease. On physical examination, the patient appeared icteric. His vital signs were as follows: temperature 37.8°C, heart rate 90 bpm, blood pressure 120/80 mmHg, and respiratory rate 16 breaths/min. Abdominal examination revealed tenderness in the right upper quadrant and a positive Murphy's sign. No other significant findings were noted on physical examination. Complete blood count showed a white blood cell count of 12,000/μL with a left shift, suggestive of an inflammatory process. Liver function tests revealed elevated total bilirubin (23 mg/dL), direct bilirubin (15.9 mg/dL), aspartate aminotransferase (AST) (632 U/L), alanine aminotransferase (ALT) (180 U/L), and gamma-glutamyl transferase (GGT) (118 U/L). These findings indicated cholestasis and hepatocellular injury. Other laboratory tests, including electrolytes, renal function, and coagulation profile, were within normal limits. Abdominal ultrasonography was performed and revealed a thickened gallbladder wall, sludge, and minimal pericholecystic fluid. There was no evidence of biliary dilatation or gallstones. Other abdominal organs were visualized and appeared within normal limits.

Based on the patient's clinical presentation, laboratory findings, and imaging results, a primary diagnosis of acute cholecystitis with non-obstructive jaundice was made. The differential diagnoses considered included; Choledocholithiasis: This condition involves the presence of gallstones in the common bile duct, which can cause biliary obstruction and jaundice. However, imaging studies in this case did not reveal any evidence of biliary dilatation or gallstones; Acute cholangitis: This is a serious infection of the bile ducts, often associated with biliary obstruction. While the patient presented with fever and elevated liver function tests, the absence of biliary dilatation on imaging made acute cholangitis less likely; Viral hepatitis: This refers to inflammation of the liver caused by a viral infection. However, the patient's clinical presentation and the absence of other risk factors for viral hepatitis made this diagnosis less probable; Autoimmune hepatitis: This is a chronic inflammatory condition of the liver caused by an autoimmune response. However, the patient's acute presentation and the absence of other autoimmune features made this diagnosis less likely; Drug-induced liver injury: This refers to liver damage caused by medications or other substances. However, the patient denied taking any medications, making this diagnosis less probable.

The patient was initially treated with intravenous fluids, antibiotics (ceftriaxone), and supportive care, including pain management and antiemetics. However, despite these measures, his jaundice and transaminitis persisted. On the fourth day of hospitalization, methylprednisolone (62.5 mg twice daily) was initiated due to persistent jaundice and evidence of liver and bile duct inflammation. Following the initiation of methylprednisolone, the patient's clinical and laboratory parameters improved significantly. His right upper quadrant pain and nausea subsided, and his bilirubin and transaminase levels progressively declined. After seven days of methylprednisolone therapy, the patient's symptoms

had resolved, and his liver function tests had normalized. He was discharged home with instructions for outpatient follow-up with a gastroenterologist. At one-month follow-up, the patient remained asymptomatic, and his liver function tests were normal. Abdominal ultrasonography showed resolution of cholecystitis. At six and twelve months follow-up, the patient continued to do well, with no recurrence of jaundice or abdominal pain. This case highlights the potential benefits of methylprednisolone as an adjuvant therapy for acute cholecystitis with non-obstructive jaundice. The patient's clinical and laboratory improvement following the initiation of methylprednisolone suggests that this therapy can reduce liver and bile duct inflammation, improve liver function, and promote the resolution of jaundice. While this case report provides evidence for the potential benefits of methylprednisolone in this setting, further studies are needed to confirm these findings and establish the optimal dosage and duration of therapy (Table 1).

The patient's initial treatment consisted of intravenous fluids in the form of normal saline administered at a rate of 20 drops per minute. This was to maintain hydration and electrolyte balance. He was also started on a course of ceftriaxone, a broad-spectrum antibiotic, given at a dosage of 2 grams twice daily to address the potential for bacterial infection. To prevent any gastrointestinal complications, esomeprazole, a proton pump inhibitor, was administered twice daily. Ondansetron, an antiemetic, was prescribed three times per day to manage nausea and facilitate oral intake. Additionally, curcuma was given three times per day, and ketorolac, an analgesic, was administered twice daily for pain management. On the fourth day of hospitalization, due to persistent jaundice and transaminitis, the decision was made to initiate adjuvant therapy with methylprednisolone. Methylprednisolone, a potent anti-inflammatory corticosteroid, was administered at a dosage of 62.5 mg twice daily for a duration of seven days. The rationale for this intervention was to mitigate the inflammatory process affecting the liver and bile ducts, thereby promoting the resolution of jaundice and improving liver function. The patient's response to treatment was closely monitored. Clinically, there was a notable improvement in his right upper quadrant pain and nausea. Laboratory investigations revealed a progressive decline in bilirubin and transaminase levels, indicating a positive response to the therapeutic interventions. Follow-up assessments were conducted to track the patient's progress. On day 7 of hospitalization, significant improvement in both symptoms and laboratory parameters was observed. By day 10, the patient's condition had stabilized sufficiently to allow for discharge home. He was provided with instructions for outpatient follow-up with a gastroenterologist to ensure continued monitoring and management. Long-term follow-up was also arranged to assess the patient's overall recovery and to monitor for any potential complications or recurrence of symptoms. At one month post-discharge, the patient reported complete resolution of symptoms, and his liver function tests had returned to normal. Abdominal ultrasonography confirmed the resolution of cholecystitis. Subsequent follow-up visits at 6 and 12 months revealed continued good health, with no evidence of gallbladder disease or recurrence of jaundice or abdominal pain. As part of the long-term management plan, the patient received recommendations for lifestyle modifications. These included dietary counseling to reduce fat intake and encourage healthier eating habits. He was also advised to abstain from alcohol consumption to promote liver health and prevent future complications. Additionally, periodic monitoring was advised, including liver function tests and abdominal imaging as needed, to ensure the continued well-being of the patient (Table 2).

Table 1. Summary of patient presentation and clinical findings.

Feature	Details
Demographics	
Age	25 years
Gender	Male
Presenting complaints	
Right upper quadrant abdominal pain	Constant, stabbing, 3-week duration, worsened over the past 2 days, aggravated by movement
Jaundice	3-week duration, yellowing of skin and sclera
Nausea	Present, accompanied by decreased appetite
Gray-colored stools	Present
Dark urine	Present, described as "tea-colored"
History	
Past medical history	No significant past medical history
Medications	None
Allergies	None
Social history	Regular alcohol consumption
Family history	No family history of liver or gallbladder disease
Physical examination	
General appearance	Icteric
Vital signs	Temperature 37.8°C, Heart rate 90 bpm, Blood pressure 120/80 mmHg, Respiratory rate 16 breaths/min
Abdominal examination	Tenderness in the right upper quadrant, positive Murphy's sign
Other	No other significant findings
Laboratory investigations	
Complete blood count	White blood cell count 12,000/μL with left shift
Liver function tests	Total bilirubin 23 mg/dL, Direct bilirubin 15.9 mg/dL, AST 632 U/L, ALT 180 U/L, GGT 118 U/L
Other	Normal electrolytes, renal function, and coagulation profile
Imaging	
Abdominal ultrasonography	Thickened gallbladder wall, sludge, minimal pericholecystic fluid, no biliary dilatation or gallstones (Figure 2)
Clinical diagnosis	
Primary diagnosis	Acute cholecystitis with non-obstructive jaundice
Differential diagnoses	Choledocholithiasis, acute cholangitis, viral hepatitis, autoimmune hepatitis, drug-induced liver injury

Table 2. Treatment and follow-up.

Phase	Details
Initial treatment	
Intravenous fluids	Normal saline at 20 drops per minute
Antibiotics	Ceftriaxone 2 grams twice daily
Proton pump inhibitor	Esomeprazole twice daily
Antiemetic	Ondansetron three times per day
Curcuma	Three times per day
Analgesic	Ketorolac twice daily
Adjuvant therapy	
Steroid	Methylprednisolone 62.5 mg twice daily, initiated on day 4 of hospitalization, continued for 7 days
Response to treatment	
Clinical	Improvement in right upper quadrant pain and nausea
Laboratory	Progressive decline in bilirubin and transaminase levels (see Figure 1)
Follow-up	
Day 7	Significant improvement in symptoms and laboratory parameters
Day 10	Discharged home with instructions for outpatient follow-up with a gastroenterologist
Long-term follow-up	
1 month	Complete resolution of symptoms, normal liver function tests, abdominal ultrasonography shows resolution of cholecystitis
6 months	Remains asymptomatic, no recurrence of jaundice or abdominal pain
12 months	Continued good health, no evidence of gallbladder disease
Recommendations	
Lifestyle modifications	Dietary counseling to reduce fat intake, alcohol cessation
Monitoring	Periodic liver function tests and abdominal imaging as needed

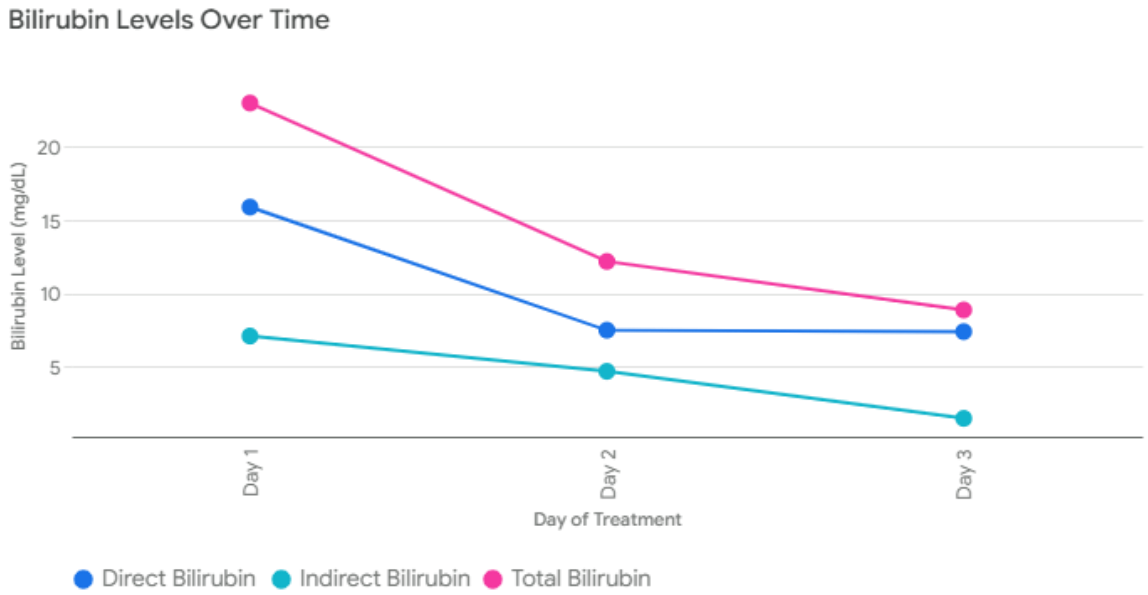

Figure 1. The changes in bilirubin levels (Total, Direct, and Indirect) over three days of treatment.

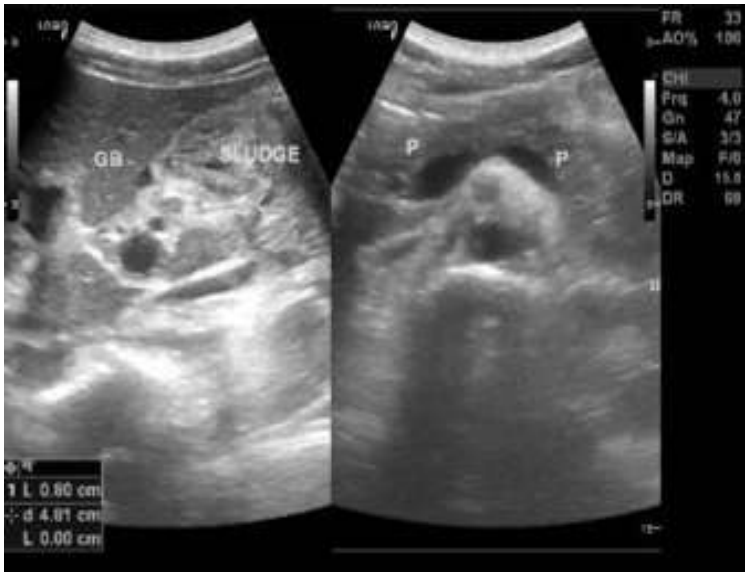

Figure 2. Abdominal USG showed cholecystitis with thickened gallbladder wall and sludge. There was minimal free fluid in the paravesical space, but no gallstones or masses were found. Other abdominal organs were within normal limits.

3. Discussion

Acute cholecystitis, a common inflammatory condition of the gallbladder, is primarily caused by gallstones or infection. The classic presentation includes right upper quadrant pain, fever, nausea, and vomiting. Jaundice, a yellow discoloration of the skin and sclera due to elevated bilirubin levels, is often associated with acute cholecystitis. In most cases, jaundice in acute cholecystitis results from common bile duct obstruction by gallstones, leading to impaired

bile flow and bilirubin accumulation. However, jaundice can also occur in acute cholecystitis without evidence of biliary obstruction, a condition known as non-obstructive jaundice. The pathogenesis of non-obstructive jaundice in acute cholecystitis is complex and not fully understood. Proposed mechanisms include direct inflammation of the liver and bile ducts, systemic inflammation affecting bilirubin metabolism, and sepsis-induced cholestasis. The management of acute cholecystitis with non-obstructive jaundice can be challenging. While cholecystectomy remains the definitive treatment for acute cholecystitis, the role of adjuvant therapies, such as steroids, is less clear. Steroids have potent anti-inflammatory effects and have been used in various liver diseases to reduce inflammation and improve liver function. However, their use in acute cholecystitis with non-obstructive jaundice has not been extensively studied. The presence of jaundice in a patient with acute cholecystitis typically raises concerns about biliary obstruction, usually caused by gallstones lodged in the common bile duct (choledocholithiasis). This obstruction impedes the flow of bile, leading to the accumulation of bilirubin in the bloodstream and subsequent jaundice. However, when jaundice occurs in the absence of any evidence of biliary obstruction on imaging studies, it presents a diagnostic challenge for clinicians. Non-obstructive jaundice in acute cholecystitis can be attributed to several factors, making the diagnostic process more complex. The inflammatory process associated with acute cholecystitis can extend beyond the gallbladder, affecting the adjacent liver parenchyma and bile ducts. This inflammation can disrupt liver function and bile flow, leading to jaundice. The close proximity of the liver and bile ducts to the gallbladder makes them susceptible to the spread of inflammation. The inflammatory mediators released during acute cholecystitis can directly damage liver cells and disrupt bile duct function, impairing bile flow and causing jaundice. Acute cholecystitis can trigger a systemic inflammatory response, leading to the release of cytokines and other inflammatory mediators. These mediators can impair liver function and bilirubin metabolism, resulting in jaundice. Cytokines such as tumor necrosis factor-alpha (TNF-alpha) and interleukin-6 (IL-6) have been shown to impair liver function and bilirubin metabolism. These cytokines can disrupt the uptake, conjugation, and excretion of bilirubin by the liver, leading to its accumulation in the bloodstream and subsequent jaundice. In severe cases of acute cholecystitis, bacterial infection can lead to sepsis, a life-threatening condition characterized by a dysregulated immune response. Sepsis can cause cholestasis, a condition in which bile flow is impaired, leading to jaundice. Sepsis can lead to a decrease in blood flow to the liver, impairing its function and ability to process bilirubin. Additionally, sepsis can cause direct damage to liver cells, further disrupting bile flow and bilirubin metabolism. The diagnostic approach to non-obstructive jaundice in acute cholecystitis involves a comprehensive evaluation to identify the underlying cause and rule out other potential etiologies. A thorough history, including the patient's presenting symptoms, past medical history, medications, and social habits, can provide valuable clues. Physical examination should focus on assessing for signs of jaundice, right upper quadrant tenderness, and other relevant findings. A detailed history can help identify risk factors for acute cholecystitis, such as gallstones, previous episodes of cholecystitis, and family history. Physical examination can reveal signs of jaundice, such as yellowing of the skin and sclera, as well as right upper quadrant tenderness, which is a hallmark of acute cholecystitis. Blood tests, including liver function tests, complete blood count, and inflammatory markers, can help assess the severity of inflammation and liver function. Elevated transaminases (AST and ALT) and gamma-glutamyl transferase (GGT) may suggest liver and bile duct inflammation. Liver function tests can reveal elevated levels of bilirubin, indicating jaundice.

Elevated transaminases (AST and ALT) and gamma-glutamyl transferase (GGT) may suggest liver and bile duct inflammation. Complete blood count can show leukocytosis, a sign of infection. Inflammatory markers, such as C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR), can also be elevated in acute cholecystitis. Abdominal ultrasonography is the initial imaging modality of choice for evaluating acute cholecystitis. It can confirm the diagnosis of acute cholecystitis and rule out biliary obstruction. If ultrasonography is inconclusive, other imaging modalities, such as computed tomography (CT) scan or magnetic resonance cholangiopancreatography (MRCP), may be considered. Ultrasonography can visualize the gallbladder and assess for signs of inflammation, such as wall thickening, pericholecystic fluid, and gallstones. It can also rule out biliary obstruction by visualizing the common bile duct and assessing for the presence of gallstones. CT scan and MRCP can provide more detailed images of the biliary system and surrounding structures, helping to identify any abnormalities that may be contributing to jaundice. In some cases, additional investigations may be necessary to rule out other potential causes of jaundice, such as viral hepatitis, autoimmune hepatitis, or drug-induced liver injury. These investigations may include serological tests for viral hepatitis, autoimmune markers, and drug screening. Serological tests can detect the presence of viral hepatitis markers, such as hepatitis B surface antigen (HBsAg) and hepatitis C antibody (anti-HCV). Autoimmune markers, such as antinuclear antibodies (ANA) and anti-smooth muscle antibodies (ASMA), can be used to assess for autoimmune hepatitis. Drug screening can identify medications or other substances that may be causing liver injury. The management of non-obstructive jaundice in acute cholecystitis focuses on addressing the underlying inflammation and providing supportive care. While cholecystectomy remains the definitive treatment for acute cholecystitis, the role of adjuvant therapies, such as steroids, is less clear. Cholecystectomy, the surgical removal of the gallbladder, is the definitive treatment for acute cholecystitis. In cases of non-obstructive jaundice, cholecystectomy can help resolve the inflammation and improve liver function. Cholecystectomy can be performed laparoscopically or open, depending on the severity of the condition and the patient's overall health. Laparoscopic cholecystectomy is the preferred approach, as it is less invasive and associated with faster recovery times. Supportive care measures, including intravenous fluids, pain management, and antiemetics, are essential in managing acute cholecystitis. These measures help maintain hydration, alleviate symptoms, and promote recovery. Intravenous fluids help maintain hydration and electrolyte balance, which can be disrupted due to nausea and vomiting. Pain management is crucial to alleviate the patient's discomfort and improve their overall well-being. Antiemetics can help control nausea and vomiting, allowing for better oral intake and preventing dehydration. The role of adjuvant therapies, such as steroids, in the management of non-obstructive jaundice in acute cholecystitis is not well established. Steroids have potent anti-inflammatory effects and have been used in various liver diseases to reduce inflammation and improve liver function. However, their use in acute cholecystitis with non-obstructive jaundice has not been extensively studied. Steroids may be considered in cases of severe inflammation or when other treatment options have failed. However, their use should be carefully weighed against the potential risks, such as increased risk of infection and delayed wound healing.[11-13]

This case report describes a patient with acute cholecystitis and non-obstructive jaundice who demonstrated a significant clinical and laboratory improvement following treatment with methylprednisolone, a synthetic glucocorticoid with potent anti-inflammatory properties. The patient

presented with a diagnostic challenge, as jaundice is typically associated with biliary obstruction in acute cholecystitis. However, imaging studies ruled out biliary obstruction, suggesting an alternative mechanism for the patient's jaundice. Non-obstructive jaundice in acute cholecystitis can be attributed to several factors, including direct inflammation of the liver and bile ducts, systemic inflammation affecting bilirubin metabolism, and sepsis-induced cholestasis. In this case, the elevated transaminase levels and GGT suggested liver and bile duct inflammation as the likely cause of the patient's jaundice. The use of methylprednisolone in this case was based on its potent anti-inflammatory effects. Glucocorticoids, such as methylprednisolone, suppress the immune response by inhibiting the production of inflammatory cytokines and chemokines. This anti-inflammatory action can reduce liver and bile duct inflammation, improve liver function, and promote the resolution of jaundice. The patient's clinical and laboratory improvement following the initiation of methylprednisolone supports the potential benefits of this therapy in acute cholecystitis with non-obstructive jaundice. The decrease in bilirubin and transaminase levels suggests a reduction in liver and bile duct inflammation, leading to improved liver function and resolution of jaundice. While this case report provides evidence for the potential benefits of methylprednisolone in acute cholecystitis with non-obstructive jaundice, further studies are needed to confirm these findings. Randomized controlled trials comparing methylprednisolone to placebo or other therapies are necessary to establish the efficacy and safety of this treatment approach. Additionally, future studies should investigate the optimal dosage and duration of methylprednisolone therapy in this setting. Methylprednisolone, a synthetic glucocorticoid, has emerged as a potential game-changer in the management of acute cholecystitis with non-obstructive jaundice. Its potent anti-inflammatory properties make it an attractive therapeutic option for this condition, which is often characterized by significant inflammation in the liver and bile ducts. This inflammation can extend beyond the gallbladder, affecting the adjacent liver parenchyma and bile ducts, leading to impaired liver function, disrupted bile flow, and jaundice. Methylprednisolone inhibits the production of inflammatory cytokines and chemokines, which are key mediators of the inflammatory response in acute cholecystitis. By suppressing the production of these mediators, methylprednisolone can reduce inflammation in the liver and bile ducts, improving bile flow and bilirubin metabolism. This reduction in inflammation helps alleviate the symptoms of acute cholecystitis, such as pain, fever, and nausea. Methylprednisolone can improve liver function by reducing inflammation and promoting hepatocyte regeneration. Inflammation can impair liver function, leading to jaundice and other complications. By reducing inflammation, methylprednisolone can help restore liver function and promote healing. This improvement in liver function can lead to a decrease in bilirubin levels and resolution of jaundice. Methylprednisolone also modulates the immune response, which can be beneficial in acute cholecystitis, especially in cases where sepsis is a concern. By suppressing the immune response, methylprednisolone can help prevent the excessive release of inflammatory mediators that can lead to sepsis and its complications. Methylprednisolone has been shown to protect liver cells from damage caused by inflammation and oxidative stress. This protection can help preserve liver function and prevent further complications. While this case report provides evidence for the potential benefits of methylprednisolone in acute cholecystitis with non-obstructive jaundice, further studies are needed to confirm these findings. However, several other studies have also suggested the potential benefits of corticosteroids, including methylprednisolone, in the management of acute cholecystitis. Corticosteroids have been shown to reduce inflammation and pain in

acute cholecystitis. A study published in the World Journal of Gastroenterology found that patients with acute cholecystitis who received corticosteroids had significantly lower pain scores and required less analgesia compared to those who did not receive corticosteroids. Corticosteroids have also been shown to improve liver function in acute cholecystitis. A study published in the Journal of Hepatology found that patients with acute cholecystitis who received corticosteroids had significantly lower levels of liver enzymes and bilirubin compared to those who did not receive corticosteroids. Corticosteroids may also help prevent complications of acute cholecystitis, such as gangrene, perforation, and pancreatitis. A study published in the British Journal of Surgery found that patients with acute cholecystitis who received corticosteroids had a significantly lower risk of developing these complications compared to those who did not receive corticosteroids. While methylprednisolone can be a valuable adjuvant therapy for acute cholecystitis, it is essential to consider its potential side effects and contraindications. Common side effects of methylprednisolone include increased appetite, weight gain, mood changes, insomnia, and fluid retention. More serious side effects, such as osteoporosis, peptic ulcers, and infections, can also occur, especially with long-term use. Methylprednisolone should be used with caution in patients with diabetes, hypertension, peptic ulcer disease, and infections. It is also contraindicated in patients with a history of hypersensitivity to corticosteroids.[14-16]

Non-obstructive jaundice in acute cholecystitis is a complex and intriguing phenomenon that challenges our understanding of liver and biliary pathophysiology. Unlike the more straightforward scenario of obstructive jaundice, where a physical blockage in the bile ducts leads to bilirubin buildup, non-obstructive jaundice arises from a complex interplay of factors that disrupt the delicate balance of bilirubin metabolism and excretion. At the heart of this intricate process lies inflammation, the body's natural response to injury or infection. In acute cholecystitis, the inflammation primarily originates in the gallbladder, triggered by gallstones, bacterial infection, or a combination of both. However, this inflammation doesn't necessarily remain confined to the gallbladder. It can spread like wildfire, engulfing the neighboring liver and bile ducts, causing direct damage and disrupting their normal function. This direct assault on the liver and bile ducts is a major contributor to non-obstructive jaundice. The inflammatory mediators released during acute cholecystitis, such as cytokines, chemokines, and prostaglandins, act as potent weapons, attacking liver cells and disrupting bile duct function. These mediators can cause swelling and inflammation in the bile ducts, narrowing their passageways and impeding bile flow. They can also directly damage liver cells, impairing their ability to process and excrete bilirubin. The liver, the body's central processing unit for bilirubin, is particularly vulnerable in this inflammatory crossfire. Hepatocytes, the workhorses of the liver, are responsible for capturing bilirubin from the bloodstream, conjugating it with glucuronic acid to make it water-soluble, and excreting it into the bile. However, when inflammation strikes, hepatocytes become dysfunctional, their metabolic machinery faltering under the onslaught of inflammatory mediators. This dysfunction disrupts the delicate balance of bilirubin processing, leading to its accumulation in the bloodstream and the telltale yellow discoloration of jaundice. But the inflammatory cascade doesn't stop at the liver's doorstep. It spills over into the systemic circulation, unleashing a torrent of cytokines and other inflammatory mediators that can wreak havoc throughout the body. These mediators can further impair liver function and disrupt bilirubin metabolism, exacerbating the jaundice. They can also trigger a cascade of metabolic derangements, such as a decrease in albumin levels, which can impair the transport of bilirubin in the

bloodstream, and an increase in bilirubin production, further fueling the jaundice. In severe cases of acute cholecystitis, the inflammatory battle can escalate into a full-blown war, with bacteria breaching the gallbladder's defenses and invading the bloodstream. This bacterial invasion can trigger sepsis, a life-threatening condition characterized by a dysregulated immune response. Sepsis can further compromise liver function and bile flow, leading to a more profound and potentially life-threatening jaundice. Sepsis can cause a decrease in blood flow to the liver, depriving it of the oxygen and nutrients it needs to function properly. This decrease in blood flow can be caused by a combination of factors, including hypotension, decreased cardiac output, and microvascular dysfunction. Sepsis can also cause direct damage to liver cells, further disrupting bile flow and bilirubin metabolism. This damage can be caused by the release of toxins from bacteria, as well as by the activation of immune cells that can attack liver cells. In addition to these direct effects on the liver, sepsis can also cause cholestasis, a condition in which bile flow is impaired. Cholestasis can be caused by several factors, including decreased bile production, impaired bile secretion, and bile duct obstruction. The combination of decreased liver blood flow, direct liver damage, and cholestasis can lead to a significant buildup of bilirubin in the bloodstream, resulting in severe jaundice.[17-18]

Methylprednisolone, a synthetic glucocorticoid, has emerged as a promising therapeutic agent in the management of various inflammatory conditions, including acute cholecystitis with non-obstructive jaundice. Its potent anti-inflammatory effects and ability to modulate the immune response make it an attractive option for addressing the complex pathophysiological mechanisms underlying this condition. At the molecular level, methylprednisolone exerts its anti-inflammatory effects by binding to glucocorticoid receptors in the cytoplasm. This binding triggers a series of intracellular events, culminating in the translocation of the receptor-ligand complex to the nucleus, where it modulates gene expression. This modulation leads to the suppression of inflammatory cytokines and chemokines, key mediators of the inflammatory response in acute cholecystitis. By suppressing the production of these mediators, methylprednisolone can effectively dampen the inflammatory cascade, reducing inflammation in the liver and bile ducts, improving bile flow, and promoting the resolution of jaundice. Beyond its anti-inflammatory effects, methylprednisolone also demonstrates remarkable hepatoprotective properties. It can improve liver function by reducing inflammation and promoting hepatocyte regeneration. Inflammation can impair liver function, leading to jaundice and other complications. By reducing inflammation, methylprednisolone can help restore liver function and promote healing. This improvement in liver function can lead to a decrease in bilirubin levels and resolution of jaundice. The hepatoprotective effects of methylprednisolone are not limited to its anti-inflammatory actions. It also modulates the immune response, which can be beneficial in acute cholecystitis, especially in cases where sepsis is a concern. By suppressing the immune response, methylprednisolone can help prevent the excessive release of inflammatory mediators that can lead to sepsis and its complications. Furthermore, methylprednisolone has been shown to protect liver cells from damage caused by inflammation and oxidative stress. This protection can help preserve liver function and prevent further complications. Oxidative stress, a condition characterized by an imbalance between the production of reactive oxygen species and the body's ability to detoxify them, can damage liver cells and contribute to liver dysfunction. Methylprednisolone's antioxidant properties can help mitigate oxidative stress and protect liver cells from damage. The clinical benefits of methylprednisolone in acute cholecystitis with non-obstructive jaundice have been demonstrated in several studies. Corticosteroids,

including methylprednisolone, have been shown to reduce inflammation and pain, improve liver function, and prevent complications such as gangrene, perforation, and pancreatitis. These findings suggest that methylprednisolone can be a valuable adjuvant therapy for this condition, especially in cases where inflammation is a significant contributor to the patient's clinical presentation. While the evidence supporting the use of methylprednisolone in acute cholecystitis is promising, it is essential to consider its potential side effects and contraindications. Common side effects of methylprednisolone include increased appetite, weight gain, mood changes, insomnia, and fluid retention. More serious side effects, such as osteoporosis, peptic ulcers, and infections, can also occur, especially with long-term use. Methylprednisolone should be used with caution in patients with diabetes, hypertension, peptic ulcer disease, and infections. It is also contraindicated in patients with a history of hypersensitivity to corticosteroids.[19-20]

4. Conclusion

This case report presents a unique case of acute cholecystitis with non-obstructive jaundice, where the patient demonstrated a remarkable response to methylprednisolone therapy. The patient's clinical and laboratory improvement following the initiation of methylprednisolone suggests that this therapy can reduce liver and bile duct inflammation, improve liver function, and promote the resolution of jaundice. While this case report provides evidence for the potential benefits of methylprednisolone in this setting, further studies are needed to confirm these findings and establish the optimal dosage and duration of therapy. The use of methylprednisolone in this case was based on its potent anti-inflammatory effects. Glucocorticoids, such as methylprednisolone, suppress the immune response by inhibiting the production of inflammatory cytokines and chemokines. This anti-inflammatory action can reduce liver and bile duct inflammation, improve liver function, and promote the resolution of jaundice. The patient's clinical and laboratory improvement following the initiation of methylprednisolone supports the potential benefits of this therapy in acute cholecystitis with non-obstructive jaundice. The decrease in bilirubin and transaminase levels suggests a reduction in liver and bile duct inflammation, leading to improved liver function and resolution of jaundice. While this case report provides evidence for the potential benefits of methylprednisolone in acute cholecystitis with non-obstructive jaundice, further studies are needed to confirm these findings. Randomized controlled trials comparing methylprednisolone to placebo or other therapies are necessary to establish the efficacy and safety of this treatment approach. Additionally, future studies should investigate the optimal dosage and duration of methylprednisolone therapy in this setting.

5. References

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