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Gall Bladder Perforation: A Presentation with **Acalculus Cholecystits**

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Abstract: Introduction: Gallbladder perforation and biliary peritonitis represent severe complications that can arise from acute cholecystitis. These complications pose significant risks to patients, leading to increased levels of illness and even death if not addressed promptly. Studies have shown varying rates of occurrence: one study found that 3.3% of 386 patients with acute cholecystitis experienced gallbladder perforation, while another reported a 5.9% incidence among 31 patients. Additionally, cases of gallbladder perforation have been documented in the literature, albeit rarely, in association with acute acalculous cholecystitis. Case Report: We present a case report of 55 years old female with complain of pain abdomen. Cect Abdomen was suggestive of Acalculus cholecystitis with possible rent perforation and adjacent mild ascites. Patient underwent open cholecystectomy and found Gall bladder rent near neck for which cholecystectomy done. Patient discharged with full recovery.

Keywords: Gall bladder perforation, Cholecystitis, Peritonitis, Cholecystectomy

1. Introduction

Acute cholecystitis is characterized by sudden biliary pain, fever, tenderness in the right upper abdomen, guarding, symptoms lasting more than 24 hours, and an elevated white blood cell count. About 90% of cases are linked to gallstones. The condition can be categorized into three distinct types based on their clinical or pathological features: acute calculous cholecystitis, acute acalculous cholecystitis, and emphysematous cholecystitis.

Acute Acalculus Cholecystitis: This rare but clinically significant condition is observed in about 5% of patients undergoing cholecystectomy. It mostly affects individuals with other clinical or pathological conditions such as trauma, non-biliary surgeries, sepsis, burns, parenteral nutrition, mechanical ventilation, multiple blood transfusions, and the use of narcotics or antibiotics. However, it can also occur in patients without these risk factors. The exact cause of acalculous cholecystitis is not completely understood but is believed to be multifactorial. Increased bile viscosity due to stasis and subsequent obstruction of the cystic duct may link acalculous cholecystitis to factors like fasting, narcotic use, dehydration, or anesthesia, all of which can contribute to bile stasis. Mucosal ischemia is also a significant factor, especially in patients with pre-existing cardiovascular conditions or those who develop the condition after trauma, sepsis, or surgery. Ischemia resulting from multiple subserosal venous and arterial thromboses of various causes may contribute to acute acalculous cholecystitis.

There are no distinct clinical differences between acute calculous and acalculous cholecystitis. However, acute acalculous cholecystitis tends to be more common in males and is associated with a higher incidence of complications and overall mortality. Patients at higher risk of severe complications are typically older and have elevated white blood cell counts. Histologically, acute acalculous cholecystitis often shows more pronounced bile infiltration, leukocyte accumulation in blood vessels, and lymphatic dilation compared to stone-associated cholecystitis, though there is significant overlap in the morphological features.

Gall Bladder Perforation: Gallbladder perforation (GBP) but potentially uncommon life-threatening complication of cholecystitis, whether calculous or acalculous. The symptoms of GBP may closely resemble those of uncomplicated acute cholecystitis, or they may present in a way that confuses the clinician. The clinical manifestations of GBP can range from peritonitis to symptoms typical of acute cholecystitis. The mortality rate for GBP is reported to be between 12% and 16%. GBP can occur in 6-12% of cases of acute cholecystitis, whether calculous or acalculous. Various classifications for GBP exist, but the Niemeier classification is the most widely used. Proposed in 1934, Niemeier's classification divides GBP into three types: type 1 (acute), characterized by generalized peritonitis; type 2 (subacute), marked by localized fluid at the perforation site with a pericholecystic abscess; and type 3 (chronic), involving internal (bilioenteric) or external (cholecystocutaneous) fistulas.

It is infrequently identified before surgery, but with a heightened level of suspicion and the use of diagnostic tools like ultrasound and CT scans, it can sometimes be detected preoperatively. Nevertheless, a conclusive diagnosis is typically made during laparotomy. Delays in establishing a definitive diagnosis are associated with higher rates of morbidity and mortality.

2. Case Report

A 55-years-old Female, with known case of hypertension (not taking medication), presented to the emergency department at MDM hospital jodhpur with a 3 day history of sudden-onset and severe abdominal pain along with abdominal distension. The pain was acute in onset, nonprogressive, dull aching in character. The pain was initially

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localized to the right upper quadrant and right lumbar region but had evolved to a more generalized distribution over time. The pain was aggravated on movement and not relieved by medications. There were no other associated symptoms. She had no past surgical history.

The patient was in obvious anxiety and distress when first encountered in the emergency room. Her vital signs were pulse of 115 beats per minute blood pressure 124/86 mm of His abdomen was distended with generalized tenderness and localised guarding in right hypochondrium on palpation. No organomegaly was appreciated on the physical examination. His bowel sounds were sluggish.

Lab Investigations

Hb- 11.6 g/dl TLC- 5.21*103/ul PLT-178*103/UL

V/M- NON REACTIVE

LFT- WNL

RFT- WNL

SE - 133/3.23

S AMYLASE- 159 IU/L

S LIPASE - 395 IU/L

Radiographic investigations

Xray FPA (fig 1)	Grossly normal (No free air under diaphragm)
USG whole	GB distended with mild pericholicystic free fluid however no e/o calculi or GB wall thickening.
abdomen	There is evidence of collection noted in hepatorenal pouch measuring 4.8x1.6 cm likely s/o infective etiology.
	Over distended GB with thin enhancing wall and surrounding pericholecystic edema. There is possible rent noted in
CECT abdomen	GB fundus measuring ~ 10mm with fluid noted in right sub phrenic space traversing to infra hepatic, right paracolic
pelvis (fig 2)	gutter extending till the pelvis. No evidence of any radiodense calculi.
	Findings suggestive of Acalculus cholecystitis with possible rent perforation and adjacent mild ascites.



Figure 1



Figure 2

After taking written consent patient planned for emergency open cholecystectomy. Patient underwent surgery through right subcoastal incision given and ~500 ml bilious fluid drained. The omentum and colon was adherent to gall bladder, adhesiolysis done by blunt and sharp dissection.

There was a rent at fundus of gall baldder of 1x1 cm noted. Cholecystectomy was done and complete hemostatsis achieved. Peritoneal lavage done with warm normal saline and abdominal drain of 28 and 32 no. Placed in subhepatic and pelvis respectively. Closure was done using loop polyamide and ethilon suture. Patient started orally on day 2. Post operative period uneventful and drain was removed on day 3. Patient discharged on POD-6 with taking orally and passing flatus motion. On dischange patient advised to review in opd after 7 days.



Figure 3

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Figure 4

3. Discussion

Gallbladder perforation is an uncommon yet extremely serious occurrence. Generally, these perforations can result from trauma, medical intervention, or unknown causes. Various factors such as gallstones, infections, cancer, steroid use, diabetes, and heart disease can increase the likelihood of gallbladder perforation.

Niemeier's 1934 classification categorized free gallbladder perforation into three types. Type 1 (acute) is linked with generalized biliary peritonitis, type 2 (subacute) involves fluid localization at the perforation site, pericholecystic abscess, and localized peritonitis, while type 3 (chronic) encompasses the development of internal or external fistulas. Recent studies have indicated higher incidences of subacute (Type 2) perforations compared to other types. Our patient experienced a type 1 gallbladder perforation due to associated generalized biliary peritonitis.

In a study detailing gallbladder perforation features in 19 patients with acute cholecystitis, the average patient age was 69 years with a female-to-male ratio of 3:2. Most patients had a history suggestive of gallbladder disease and concurrent medical conditions such as cardiac, pulmonary, renal, nutritional, or metabolic diseases. Perforations typically occurred within 72 hours in these patients, although reports suggest they can occur as early as 2 days after acute cholecystitis onset or after a few weeks.

Our case is exceptional because the patient lacked prior history indicative of gallbladder disease like gallstones or malignancy and had no known medical conditions predisposing to gallbladder perforation. Histopathological features of acute-on-chronic examination revealed cholecystitis, suggesting previous clinically cholecystitis episodes. During surgery, dense adhesions around the gallbladder were observed, hinting at past episodes of acute cholecystitis, though the patient denied experiencing abdominal pain previously. The gallbladder perforation likely occurred within the initial 24-48 hours of symptom onset, near the gallbladder neck, an uncommon site compared to the more frequently reported fundus and body perforations due to poorer vascular supply.

Initially, an abdominal X-ray was performed, followed by a CT scan when no signs of pneumoperitoneum or intestinal obstruction were evident. In a series of 17 patients with gallbladder perforation presenting with acute abdomen, CT scan served as the primary radiological investigation. CT sensitivity in detecting gallbladder perforation has been reported at 88%, higher than that of ultrasonographic examination.

Early surgical intervention is crucial in managing gallbladder perforation, as delayed diagnosis and treatment initiation are associated with increased morbidity and mortality.

4. Conclusion

In summary, patients with clinical signs and risk factors for gall bladder perforation should undergo surgical intervention early in their illness to significantly reduce the associated morbidity and mortality, especially in cases of acalculous cholecystitis. While CT scans play a crucial role in detecting gall bladder perforation, a high level of clinical suspicion is essential for accurate diagnosis. Laparoscopic cholecystectomy may be considered for managing gall bladder perforation, but surgeons should be prepared to switch to an open procedure if the anatomy is unclear.

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