

ACUTE AND CHRONIC CALCULUS CHOLECYSTITIS: PATHOPHYSIOLOGY, PAIN MECHANISMS, AND SURGICAL DECISION-MAKING**Dr. Lokesh Kumar Tamta*¹, Dr. Ranjit Singh², Dr. Bhoomi Soni³**PG Scholar¹, HOD and Professor², Associate Professor³***Corresponding Author: Dr. Lokesh Kumar Tamta**

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ABSTRACT

Cholelithiasis is a common hepatobiliary disorder characterized by the formation of gallstones within the gallbladder.^[1] Obstruction of the cystic duct by gallstones leads to cholecystitis, which may present as acute or chronic inflammation.^[2] Acute calculus cholecystitis is a surgical emergency associated with severe pain, inflammation, and systemic response, whereas chronic cholecystitis represents long-standing gallstone disease with recurrent low-grade inflammation.^[3] Although laparoscopic cholecystectomy is the gold standard treatment for gallstone disease, it is often deferred or avoided in acute settings due to distorted anatomy, higher complication rates, and patient instability.^[4] This article elaborates the pathophysiology, clinical features, pain mechanisms, and surgical decision-making in acute and chronic calculus cholecystitis with evidence-based in-text references.

KEYWORDS: Cholelithiasis, Acute cholecystitis, Chronic cholecystitis, Gallstone disease, *Laparoscopic cholecystectomy*, Calot's triangle.**INTRODUCTION**

Gallstone disease affects approximately 10–20% of the adult population worldwide and is more common in elderly individuals, females, and patients with metabolic comorbidities.^[1,3] Only a subset of patients with gallstones develops symptoms or complications requiring surgical intervention.^[1,2]

Cholecystitis is classified into

- Acute calculus cholecystitis
- Chronic calculus cholecystitis

Understanding the differences between these conditions is essential for safe surgical planning and patient outcomes.

ACUTE CALCULUS CHOLELITHIASIS WITH ACUTE CHOLECYSTITIS**Pathophysiology**

Gallstones develop due to supersaturation of bile with cholesterol or pigment, combined with reduced gallbladder motility and bile stasis. Predisposing factors include altered bile composition, impaired gallbladder emptying, and mucin hypersecretion.^[1]

Obstruction of the Cystic Duct

A gallstone becomes impacted in the cystic duct or Hartmann's pouch, resulting in complete obstruction of bile outflow. This obstruction is the primary initiating event in acute calculous cholecystitis.^[4]

Gall Bladder Distention and Increased Intraluminal Pressure

Continued secretion of mucus by the gallbladder mucosa leads to progressive distension. Rising intraluminal pressure compromises venous and lymphatic drainage, causing edema of the gallbladder wall.

Ischemia and Mucosal Injury

Sustained pressure impairs arterial blood supply, resulting in ischemia of the gallbladder wall. Ischemic injury predisposes to necrosis and increases susceptibility to infection (Schwartz).

Chemical Inflammation

Concentrated bile salts cause direct chemical irritation of the mucosa. Phospholipase A converts lecithin into lysolecithin, a potent inflammatory agent that exacerbates mucosal damage.

Secondary Bacterial Infection

Bacterial colonization commonly occurs due to bile stasis. Frequently isolated organisms include *Escherichia coli*, *Klebsiella*, and *Enterococcus*. Infection intensifies inflammation and systemic inflammatory response.

Acute Inflammatory Response

Release of inflammatory mediators such as prostaglandins and cytokines leads to:

1. Gallbladder wall thickening
2. Serosal inflammation
3. Severe right upper quadrant pain
4. Fever, leukocytosis, and raised CRP

Progression to Complications (If Untreated)

1. Persistent inflammation and ischemia may result in:
2. Gangrenous cholecystitis
3. Empyema of gallbladder
4. Gallbladder perforation
5. Biliary peritonitis and sepsis

Pathophysiological Sequence (Flowchart)

GALLSTONE FORMATION → CYSTIC DUCT OBSTRUCTION → BILE STASIS → GALLBLADDER DISTENSION → ISCHEMIA → CHEMICAL + BACTERIAL INFLAMMATION → ACUTE CHOLECYSTITIS

Common organisms include *E. coli*, *Klebsiella*, and *Enterococcus* species.^[4]

Clinical Features

- Severe, continuous right upper quadrant pain
- Pain radiating to right shoulder or interscapular region
- Fever, nausea, vomiting
- Positive Murphy's sign
- Laboratory findings:
- Leukocytosis
- Raised CRP

Imaging (USG)

- Gallstones
- Gallbladder wall thickening (>3 mm)
- Pericholecystic fluid^[6]

Pain Mechanism in Acute Cholecystitis

- Pain is severe and persistent due to:
- Gallbladder distension
- Ischemia
- Serosal and peritoneal irritation
- Inflammatory mediators stimulate visceral and somatic nerve endings, causing intense pain.^[7]

CHRONIC CALCULUS CHOLELITHIASIS WITH CHRONIC CHOLECYSTITIS

Pathophysiology

Persistent gallstone formation (calculi cholelithiasis)

- Chronic cholecystitis almost always occurs in the presence of long-standing gallstones.

- Gallstones cause continuous mechanical irritation of the gallbladder mucosa and repeated minor obstruction of the cystic duct.^[1]

Repeated episodes of transient cystic duct obstruction

- Gallstones intermittently obstruct the cystic duct, leading to recurrent biliary colic.
- Obstruction is usually partial and temporary, unlike the complete obstruction seen in acute cholecystitis.

Recurrent low-grade inflammation

- Repeated obstruction and bile stasis result in chronic low-grade inflammation of the gallbladder wall.
- The inflammatory process is gradual and persistent.

Chemical mucosal injury

- Concentrated bile salts cause chronic chemical irritation of the gallbladder mucosa.
- Lecithin is converted into lysolecithin, which causes ongoing epithelial damage.

Fibrosis and thickening of gallbladder wall

Chronic inflammation leads to:

- Fibrosis of the gallbladder wall
- Muscular hypertrophy
- Loss of normal elasticity

Gallbladder contracture and functional impairment

Progressive fibrosis results in a:

- Shrunken
- Rigid
- Poorly contractile gallbladder

This causes impaired bile storage and emptying.

Mucosal atrophy and Rokitansky–Aschoff sinuses

- Chronic inflammation leads to mucosal atrophy.
- Herniation of mucosa into the muscular layer forms Rokitansky–Aschoff sinuses, a characteristic feature of chronic cholecystitis.

Calcification and porcelain gallbladder (late stage)

- Long-standing disease may cause calcium deposition in the gallbladder wall.
- Porcelain gallbladder is associated with an increased risk of gallbladder carcinoma. Minimal role of bacterial infection
- Bacterial infection is uncommon in chronic cholecystitis. Inflammation is mainly mechanical and chemical, not infective. (“burnt-out gallbladder”).^[4]

Pathophysiological Sequence (Flowchart)

LONG-STANDING GALLSTONES → INTERMITTENT CYSTIC DUCT OBSTRUCTION → BILE STASIS → REPEATED LOW-GRADE INFLAMMATION → FIBROSIS & WALL THICKENING → SHRUNKEN, NON-FUNCTIONAL GALLBLADDER

Clinical Features

- Recurrent biliary colic
- Postprandial abdominal discomfort
- Dyspepsia, bloating, fatty food intolerance
- No fever or systemic toxicity

Ultrasound findings

- Gallstones
- Thickened fibrotic gallbladder wall
- Contracted gallbladder

Pain mechanism in Chronic Cholecystitis**Pain is:**

- Intermittent
- Dull or colicky

Caused by

- Transient cystic duct obstruction
- Poor gallbladder contraction
- Absence of active inflammation explains reduced severity compared to acute disease.^[1]

WHY LAPAROSCOPIC SURGERY IS OFTEN NOT PERFORMED IN ACUTE CONDITIONS

Although early laparoscopic cholecystectomy is recommended in selected patients, it is frequently avoided or deferred in acute cholecystitis due to the following reasons:

1. Distorted Calot's Triangle Anatomy

- Acute inflammation causes:
- Edema
- Adhesions
- Obscuration of cystic duct and artery
- Increases risk of common bile duct injury.^[5]

2. Increased Risk of Intraoperative Bleeding

- Inflamed tissues are friable and bleed easily.
- Difficult hemostasis during laparoscopic dissection.

3. Severe Inflammatory and Septic State

Conditions such as:

- Empyema gallbladder
- Gangrenous cholecystitis
- Perforation with peritonitis
- Increase perioperative morbidity and mortality.^[6]

4. Poor Patient Physiological Reserve

Elderly patients or those with:

- Cardiac disease
- Renal failure
- Respiratory compromise
- May not tolerate pneumoperitoneum and prolonged anesthesia.

5. High Conversion Rate to Open Surgery

- Acute cholecystitis has a significantly higher conversion rate due to unclear anatomy and complications.^[7]

MANAGEMENT APPROACH**Acute Cholecystitis**

Initial conservative management:

- Nil per oral
- IV fluids
- Broad-spectrum antibiotics
- Analgesia

Surgical options

- Early laparoscopic cholecystectomy in stable patients
- Interval cholecystectomy after inflammation subsides
- Percutaneous cholecystostomy in high-risk patients

Chronic Cholecystitis

- Elective laparoscopic cholecystectomy is the treatment of choice
- Lower complication rates due to well-defined anatomy

CONCLUSION

- Acute and chronic calculus cholecystitis represent different pathological stages of gallstone disease.
- Acute cholecystitis is characterized by severe pain, inflammation, and higher surgical risk.
- Chronic cholecystitis presents with recurrent mild symptoms and is best managed by elective laparoscopic surgery.
- Avoidance of laparoscopic surgery in acute settings is often justified due to anatomical distortion, bleeding risk, and patient instability.
- Careful patient selection and timing of surgery are critical for optimal outcomes.

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