

❑ CARCINOMA OF GALLBLADDER AND CHOLICYSTITIS

GROUP: - 4

SUBJECT :- PATHOANATOMY

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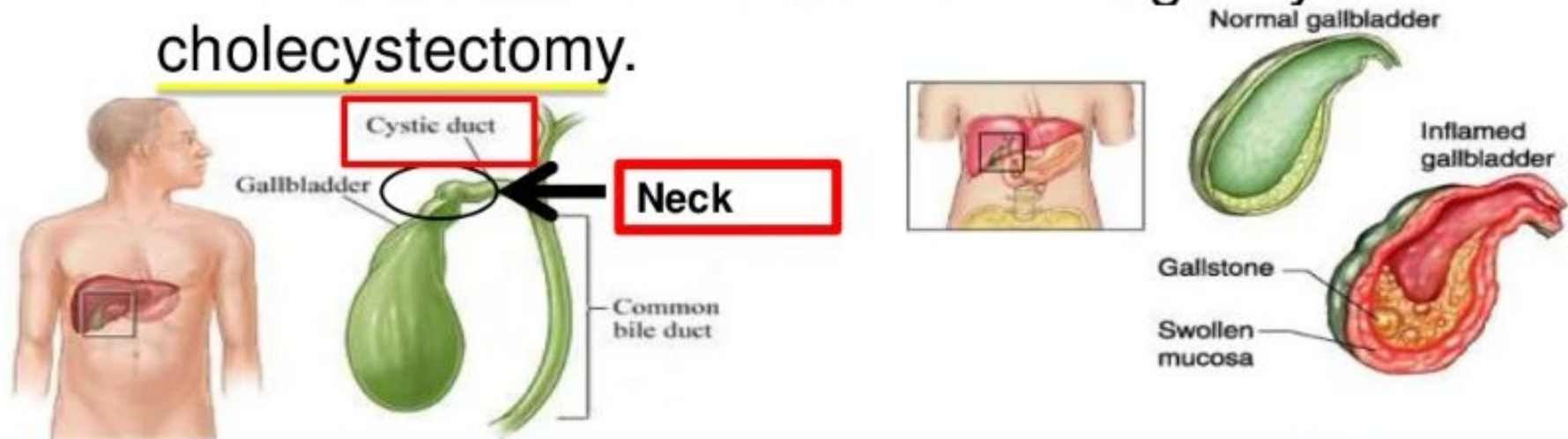
ANIKET MUSALE

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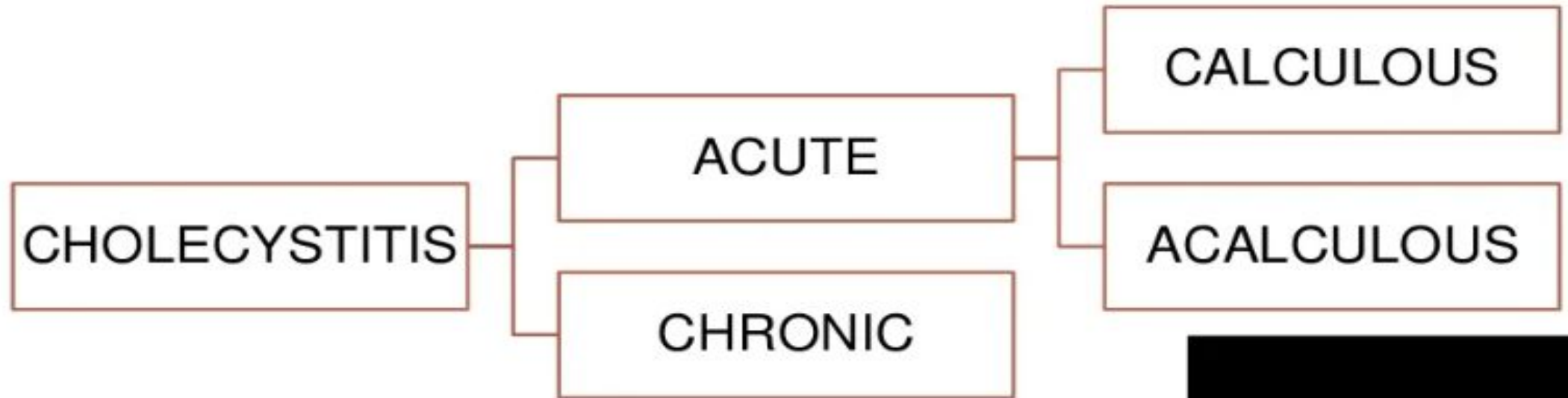
DEFINITION

Acute inflammation of gallbladder that **contains stones** and is precipitated by obstruction of the gallbladder neck or cystic duct.

- The most common major complication of gallstones.
- The most common reason for emergency cholecystectomy.





CLASSES OF CHOLECYSTITIS





PATHOGENESIS

- Acute **CALCULOUS** *cholecystitis* results from chemical irritation and inflammation of the obstructed gallbladder.
 - The action of mucosal phospholipases hydrolyzes luminal lecithins (phospholipids) to toxic **LYSOLECITHINS**.
 - The normally protective glycoprotein mucus layer is disrupted, exposing the mucosal epithelium to the direct **DETERGENT action** of bile salts.
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- **Prostaglandins** released within the wall of the distended gallbladder contribute to mucosal and mural inflammation.
 - Gallbladder **dysmotility** develops

↓
distention and increased intraluminal pressure

↓
compromise blood flow to the mucosa.

↓
ischemia

MORPHOLOGY

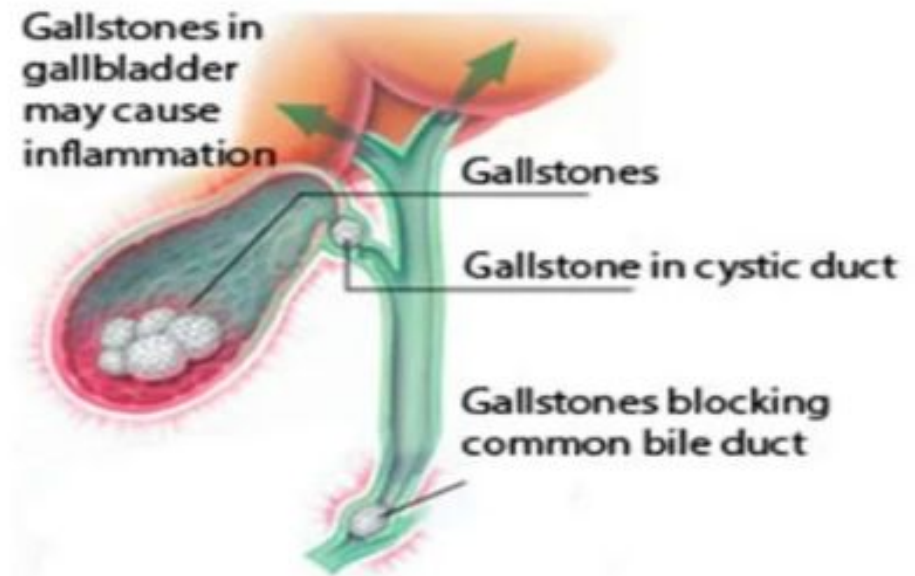
In **acute cholecystitis** basically,


- Gallbladder is usually enlarged and tense
- Characterized as a bright red or blotchy, violaceous to green-black discolouration.
- Imparted by subserosal haemorrhages.
- Serosal covering is layered by:
 - Fibrin
 - Definite suppurative



In **acute calculous cholecystitis**,

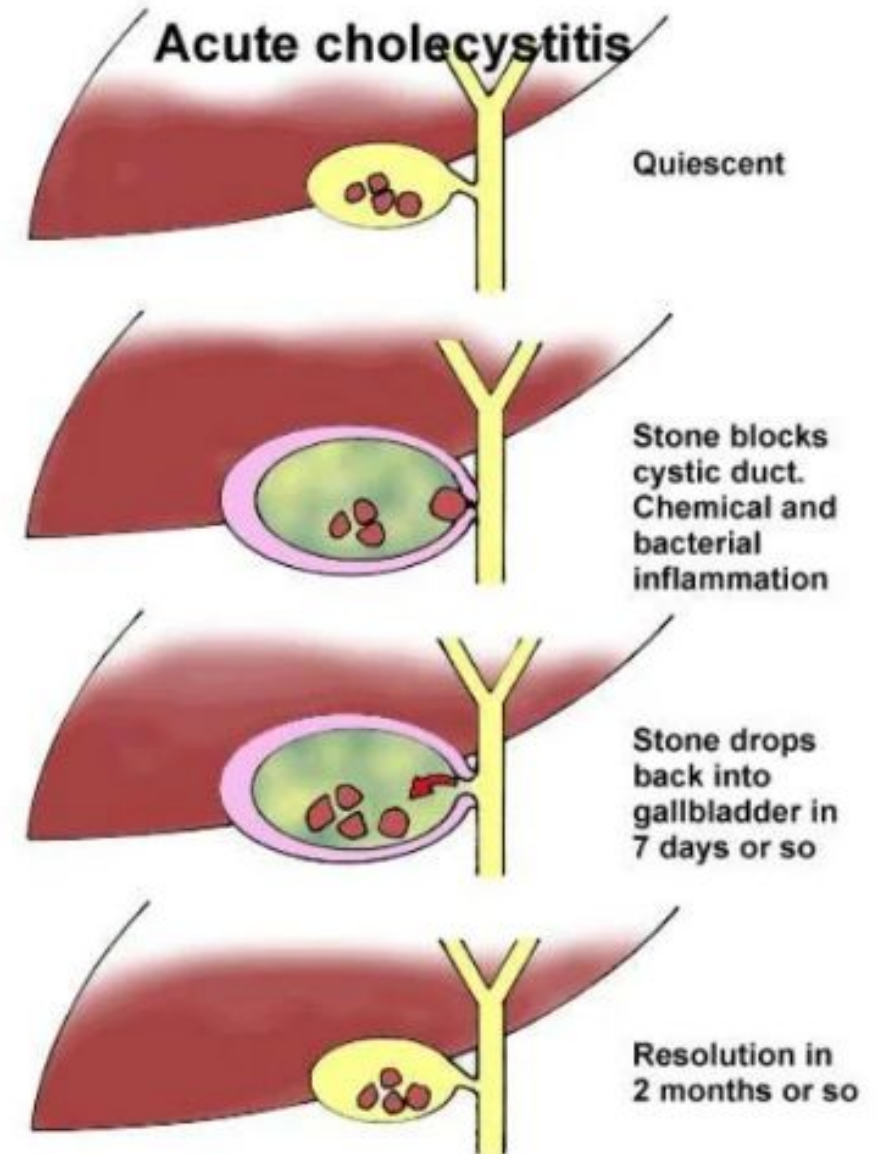
- Obstructing stones are usually present in the **neck of the gallbladder or cystic duct**.
- The gallbladder lumen may contain one or more stones, filled with cloudy or turbid bile which may contain large amounts of fibrin, pus and haemorrhage.



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- **Empyema of gallbladder** – the contained exudate is virtually pure pus.
 - In mild cases, the wall is thickened, edematous and hyperemic.
 - **Gangrenous cholecystitis** – severe, gallbladder turns into green-black necrotic organ, with small-to-large perforations.
 - **Acute emphysematous cholecystitis** – invasion of gas-forming organisms i.e., clostridia and coliforms.

CLINICAL FEATURES

- Patients usually, not always, experienced previous episodes of pain.
- May appear with remarkable suddenness & constitute an acute surgical emergency.
- Or may present with mild symptoms without medical intervention.
- Attacks usually subside in 7-10 days.
- 25% of patients progressively develop more severe symptoms
 - immediate surgical intervention.



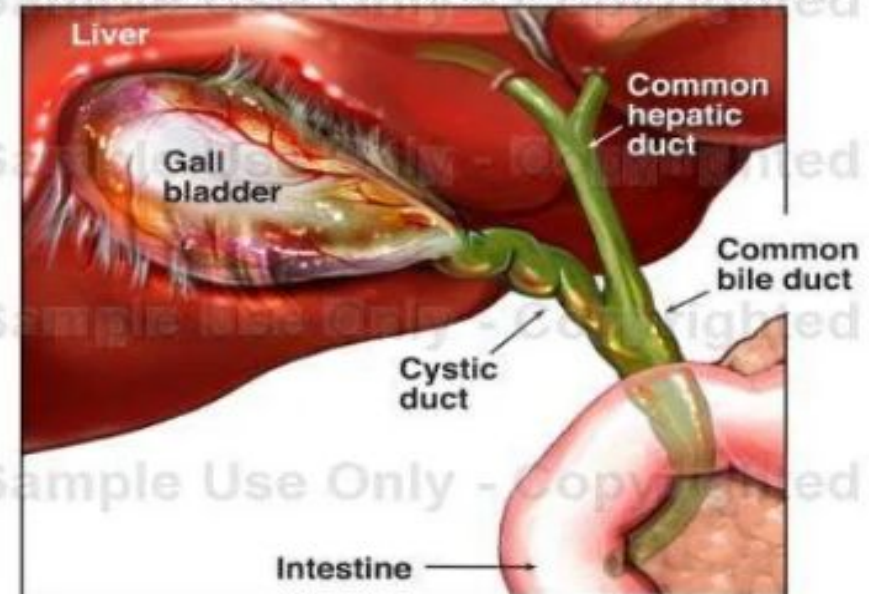
DEFINITION

Acute inflammation of gallbladder that has no relation with gallstones.

- 5%-12% of gallbladders removal contain no gallstones.
- Mostly happen in seriously ill patients.

Preoperative condition with inflamed gallbladder

Pre-operative Condition with Inflamed Gall Bladder-Cholecystitis



RISK FACTORS

- (1) **Sepsis** with hypotension and multisystem organ failure;
- (2) **Immunosuppression**
- (3) **Major trauma and burns**
- (4) **Diabetes mellitus**
- (5) **Infections**
(Salmonellosis & Cholera)

Subserosal perforation in diabetic patient with acute emphysematous cholecystitis.



note the greenish hue; this is due to a subserosal perforation in a diabetic patient with Acute Emphysematous Cholecystitis

PATHOGENESIS

Results from ischemia of cystic artery



Contributing factors:

- Inflammation & edema of the wall that compromise blood flow
- Gallbladder stasis
- Accumulation of microcrystals of cholesterol (biliary sludge)
- Viscous bile
- Gallbladder mucus



Cystic duct obstruction in the absence of frank stone formation

MORPHOLOGY

- There are no specific morphologic differences between acute acalculous and calculous cholecystitis, except for the absence of macroscopic stones in acalculous form.

CLINICAL FEATURES

- The symptoms are more **insidious** – underlying conditions.
- Higher proportion of patients – no symptoms referable to gallbladder.
- Incidence of **gangrene** and **perforation** is higher than in calculous cholecystitis.
- Rarely, acute acalculous occurs due to primary bacterial infection (e.g., *Salmonella typhi*, staphylococci).
- Less painful acute acalculous – systemic vasculitis, severe atherosclerotic ischemic disease in elderly, AIDS patients & biliary tract infection.



Gangrenous gallbladder with empyema



Perforation (hole/piercing) at the apex of gallbladder

MANAGEMENT FOR ACUTE CHOLECYSTITIS

- Initial treatment management is conservative, consisting of nil by mouth, IV fluids, opiate analgesia and **IV antibiotics (cephalosporins, fluoroquinolones or piperacillin/tazobactam)**.
- **Cholecystectomy** cures acute cholecystitis and relieves biliary pain. It is usually delayed for a few days to allow the symptoms to settle.
- Surgery may be delayed when patients have ~~an underlying severe chronic~~ disorder (eg, cardiopulmonary) that increases the surgical risks. In such patients, cholecystectomy is deferred until medical therapy stabilizes the comorbid disorders or until cholecystitis resolves.
 - If cholecystitis resolves, cholecystectomy may be done ≥ 6 wk later. Delayed surgery carries the risk of recurrent biliary complications.



CHRONIC CHOLECYSTITIS

- In most cases, it develops without any history of acute attacks, but in some cases, it happens as a **sequel to repeated bouts of acute cholecystitis**.
- Almost associated with gallstones but they do not have direct role in development of pain or inflammation.
 - Symptoms and morphologic alterations similar to those seen in calculous form.
- Since it is associated with *cholelithiasis* in more than 90% of cases, the patient populations are the same as those for gallstones.

MORPHOLOGY – GROSS

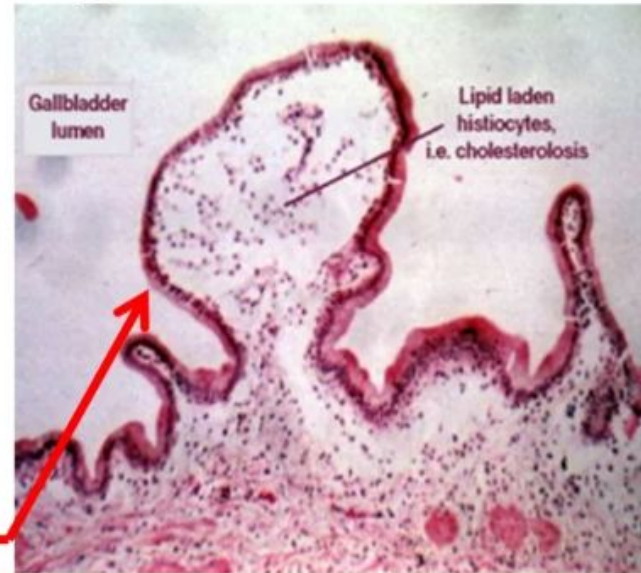
- The **serosa** - smooth and glistening but maybe dulled by subserosal fibrosis.
- **Dense fibrous adhesions.**
- Wall - **thickened** with opaque gray-white appearance.
- In uncomplicated cases – lumen contains fairly clear, green-yellow, **mucoïd bile and stones.**
- **Mucosa** is preserved.



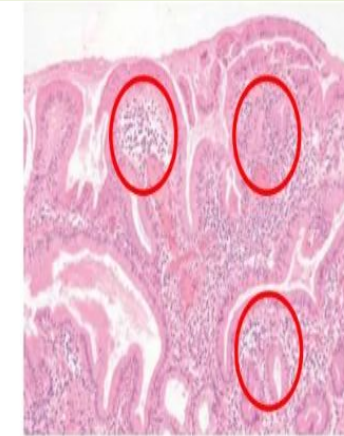
Notice thickness of galldladder wall, abundant polyhedric stones and small papillary tumor in the cystic duct.

Morphology – histologic examination

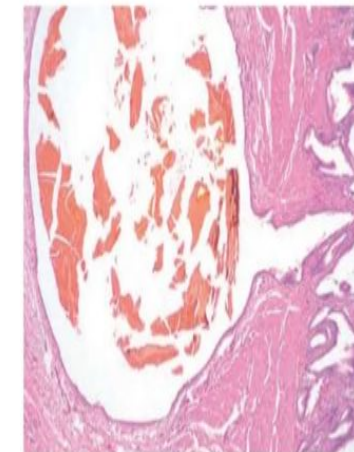
- **In the mildest cases**, only **scattered lymphocytes, plasma cells and macrophages** are found in the mucosa & subserosal fibrous tissue.
- **In advanced cases**, there is marked **subepithelial & subserosal fibrosis**, with mononuclear cell infiltration.
- **Buried crypts of epithelium** due to reactive **proliferation of mucosa & fusion of mucosal folds**.
- **Outpouchings of mucosal epithelium** through the wall – **Rokitansky-Aschoff sinuses**



Enlarged mucosal folds of the gallbladder and infiltrate of foamy histiocytes, very little inflammation, found in the muscular wall and serosal fat.



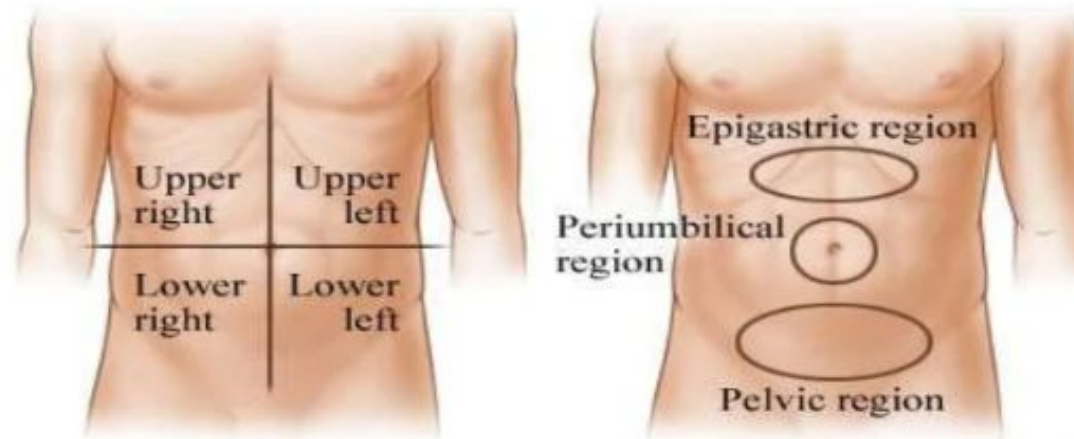
The gallbladder mucosa is infiltrated by inflammatory cells



Outpouching of the mucosa through the wall forms Rokitansky-Aschoff sinus (contains bile).

CLINICAL FEATURES

- Has no striking manifestations of acute forms.
- Usually characterized by recurrent attacks of steady **epigastric or right upper quadrant pain**.
- Nausea, vomiting and intolerance for fatty foods.



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❑ CARCINOMA OF GALLBLADDER

EPIDEMIOLOGY



More common
in women



Occurs in 7th
decade of life



Mexico & Chile
– high
incidence of
gallstone
disease



In US, most
common in
Hispanics and
Native
Americans



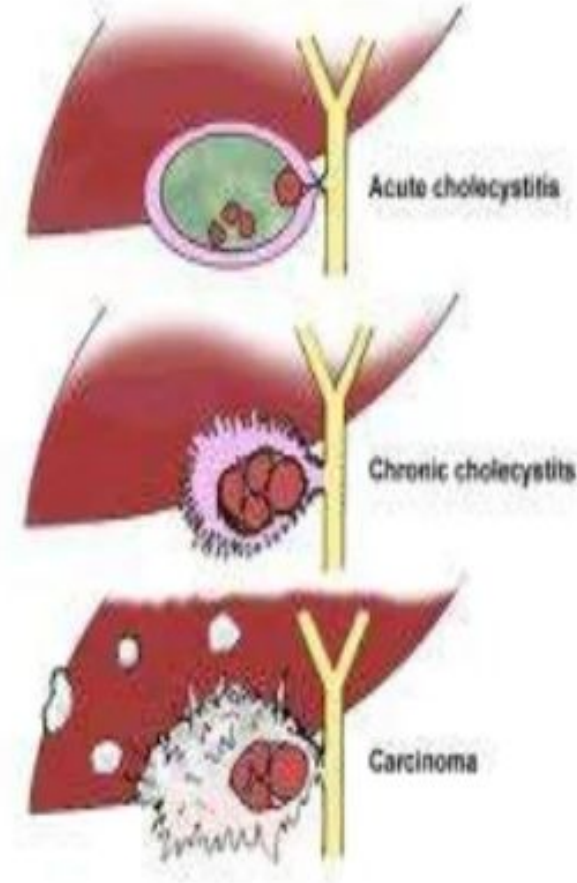
Although
uncommon, it
is the most
frequent
malignant
tumor of biliary
tract



PATHOGENESIS

- Gallbladder cancer arises in the **setting of chronic inflammation**. In the vast majority of patients (>75%), the source of this chronic inflammation is **cholesterol gallstones**.
- The presence of gallstones increases the risk of gallbladder cancer 4- to 5-fold.
- Other unusual causes are associated with gallbladder cancer, including primary sclerosing cholangitis, ulcerative colitis, liver flukes, chronic *Salmonella typhi* and paratyphi infections, and *Helicobacter* infection.

Chronic cholecystitis and carcinoma





MORPHOLOGY

Cancer may exhibit **exophytic** or **infiltrating** growth patterns.

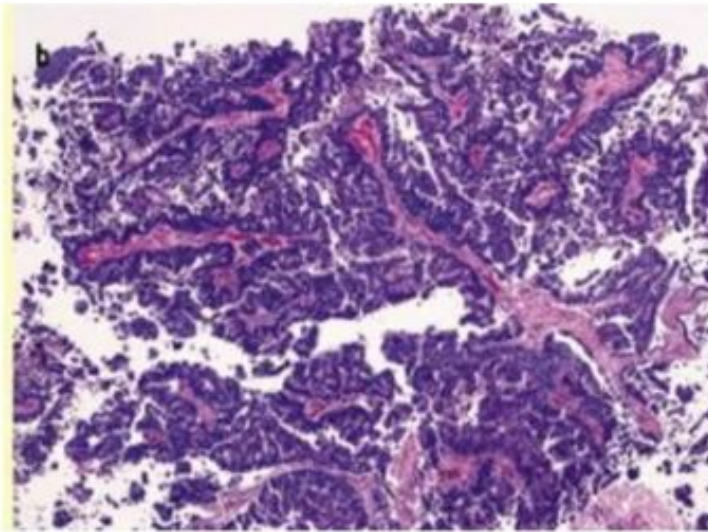
- The **infiltrating pattern** is more common and usually appears as a poorly defined area of thickening and induration of the gallbladder wall.
- Deep ulceration can cause direct penetration of gallbladder wall or fistula formation to adjacent viscera where neoplasm grow.
- These tumors are scirrhous and very firm.

- The **exophytic pattern** grows into the lumen as an irregular, cauliflower-like mass but also invades the underlying wall.
- Luminal portion may be necrotic, hemorrhagic and ulcerated.
- Most common sites: fundus & neck; 20% involve lateral walls.

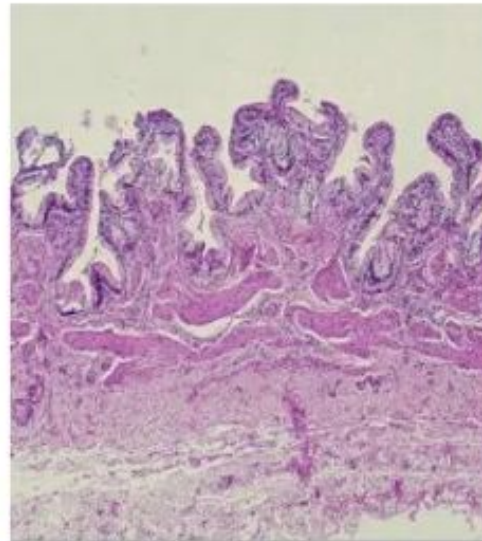


The opened gallbladder contains a large, exophytic tumor that virtually fills the lumen

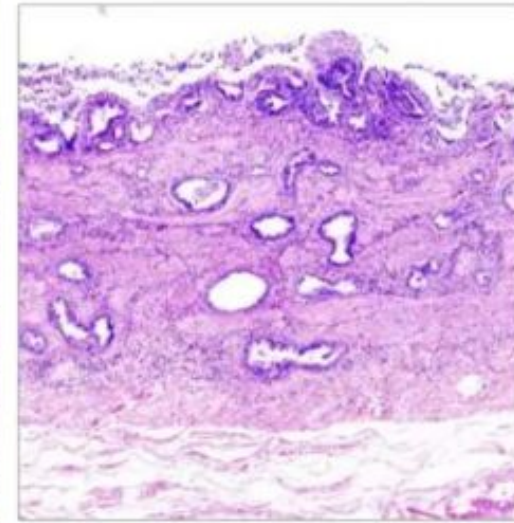
- Most are **adenocarcinomas** – may be **papillary** or poorly differentiated.
- About 5% are **squamous cell carcinomas**.
- **Neuroendocrine tumors**, which is rare, also occur.
- By the time this cancer is discovered, most have invaded the **liver** or spread to the **bile ducts** or **portal hepatic lymph nodes**.



Papillary pattern



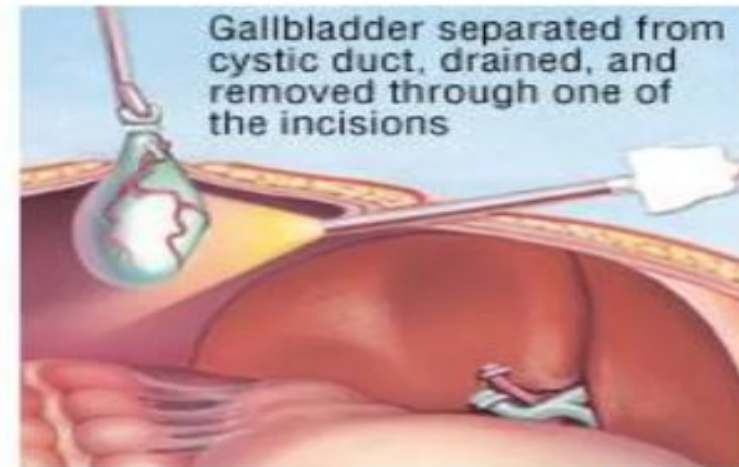
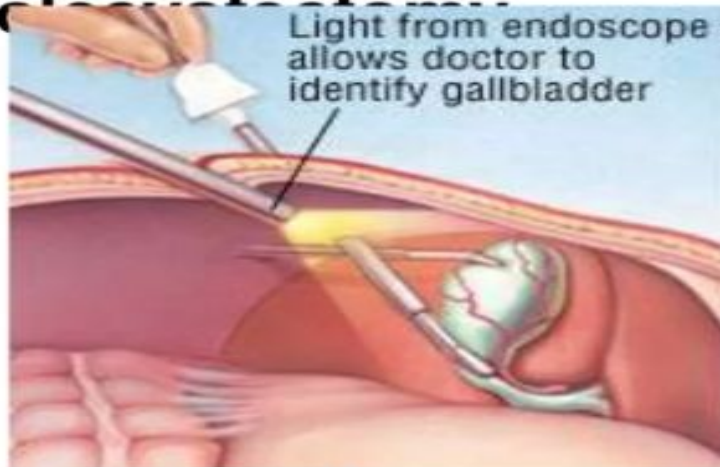
NORMAL



ADENOCARCINOMA

CLINICAL FEATURES

- Onset is **insidious** and indistinguishable from those associated with cholelithiasis (e.g., abdominal pain, jaundice, anorexia and nausea and vomiting).
- Early detection may be possible in patients with palpable gallbladder & acute cholecystitis before extension of tumor into adjacent structures.
- Or when carcinoma is found during **cholecystectomy**.



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TO MAKED
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SAVE GALLBLADDER

THANK YOU FOR YOUR ATTENTION