

The background features abstract, overlapping green geometric shapes in various shades, primarily on the left and right sides, framing a central white area.

DISORDERS OF THE GALLBLADDER

► Disorders of the gallbladder

- **Cholelithiasis** (formation of gall stones)
- **Cholecystitis** (inflammation of the gall bladder)
- **Tumors**

CHOLELITHIASIS

- Common disease affecting 10-20% of adults
- >80% are asymptomatic
- Two main types of gall stones:
 - 1) Cholesterol stones (80% in west)
 - 2) Bilirubin calcium salts (pigment) stones
- Pathogenesis:
 - 1) bile supersaturation with cholesterol
 - 2) nucleation: promoted by gallbladder hypomotility (stasis) *stasis of bile in gallbladder, promoted by 11 11*
 - 3) Cholesterol crystals remaining long enough to aggregate

Appearance

- **Cholesterol stones:** ^{NOT in bile ducts} exclusively in GB, single or multiple, multi-faceted, most are radiolucent _(can NOT be seen on Xray)
 - Pure: pale yellow
 - Mixed: gray white to black, containing calcium carbonate, phosphates & bilirubin
- **Pigment stones:** ^{NOT only in GB} anywhere in biliary tree, contain calcium salts of unconjugated bilirubin (calcium bilirubinate), mucin glycoproteins & cholesterol
 - Black: in sterile GB bile, small, numerous, friable, 50-75% are radioopaque _{can be seen on Xray}
 - Brown in infected bile ducts, single or few, soft & greasy, radiolucent

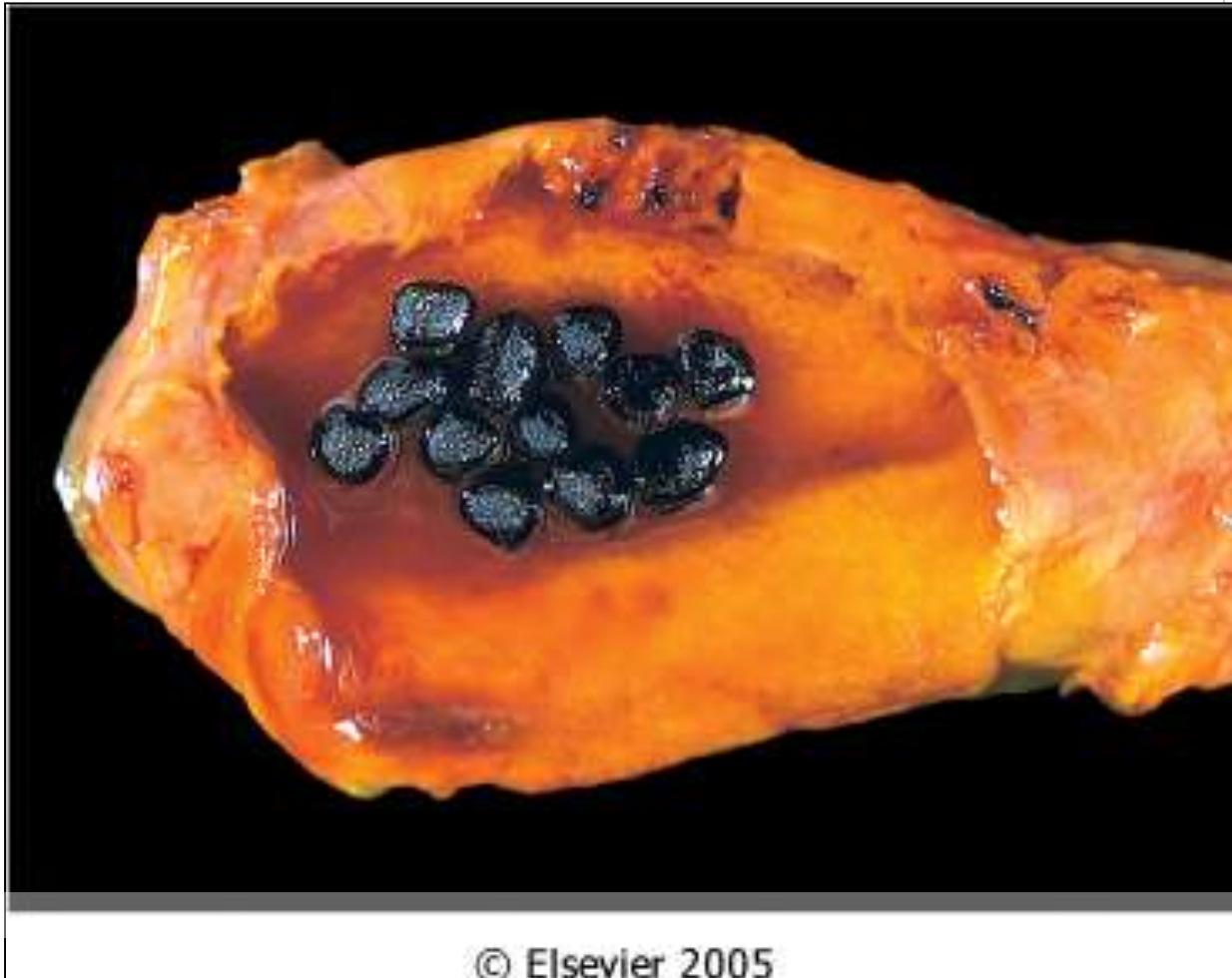
Cholesterol gallstones

characterized by yellow color especially the pure cholesterol stones, sometimes they are mixed with other solutes giving them other discolorations



Pigmented gallstones

black, numerous, friable, radiopaque, mostly formed of calcium bilirubinate



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RISK FACTORS OF CHOLELITHIASIS

- ▶ Affects 80% of people
- ▶ No identifiable risk factors other than age and gender
- ▶ **Risk factors for cholesterol stones**
 - Age: elderly > young adults ^{>40 y.}
 - Gender: females (2:1)
 - Oral contraceptives (OCPs), pregnancy
 - Demography: Western World;
 - Gallbladder stasis
 - Family history.
 - Inborn disorders of bile acid metabolism
 - Obesity
 - Hyperlipidemia
 - Rapid weight loss
 - Treatment with the hypocholesterolemic (lipids lowering drugs)

► **Risk factors for pigment stones**

- Demography: Asians, rural areas
- Chronic hemolytic syndromes *sickle cell anemia, thalassemia*
- Biliary infection
- Gastrointestinal disorders: *(malabsorption)*
 - Ileal disease, e.g. Crohn's disease
 - Ileal resection or bypass
 - Cystic fibrosis with pancreatic insufficiency *+malabsorption*

CLINICAL FEATURES OF CHOLELITHIASIS

► Clinical presentation:

- 70-80% are asymptomatic
- Biliary pain, constant or colicky from an obstructed gallbladder or biliary tree (typical acute presentation)
- Associated with inflammation of gallbladder

► Complications:

- Empyema
pus in gallbladder
- Perforation
- Fistulae induced by stones, between GB or biliary tract with other organs
- Inflammation of biliary tree (cholangitis)
- Obstructive cholestasis (jaundice)
- Pancreatitis obstruction of pancreatic ducts
- Intestinal obstruction (“gallstone ileus”) (bowel)
gallstones can escape GB cystic duct → bile ducts → Small bowel → intestinal obstruction

CHOLECYSTITIS

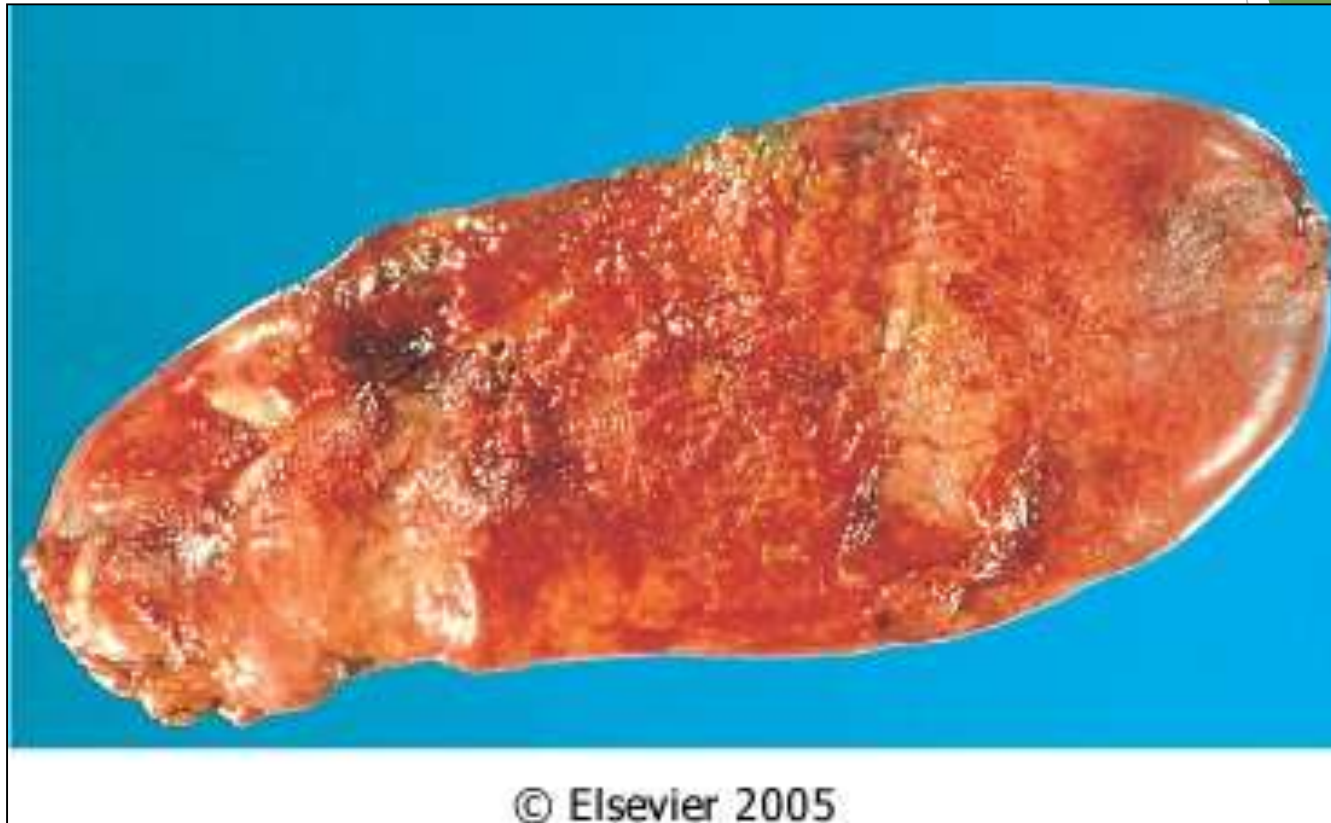
- Inflammation of the gallbladder
- **Almost always associated with gallstones**
- One of the most common indications for abdominal surgery
- Epidemiologic distribution similar to cholelithiasis *examine specimens*
- Classification: *acute / chronic*
 - Acute calculous *(related to gall stones)*
 - Acute acalculous
 - Chronic
 - Acute on top of chronic

TYPES OF ACUTE CHOLECYSTITIS

- **Acute calculous cholecystitis:** caused by obstruction of GB neck or cystic duct by stones
 - Chemical irritation & inflammation of GB wall
 - Blood flow compromise due to GB distension & pressure on blood supply
 - Symptoms may be mild or sudden & severe
- Most common reason for emergency cholecystectomy.
- Mostly in absence of bacterial infection. (bile / specimen will be sterile)
- **Acute acalculous cholecystitis:** 5-12% of cases
 - Seen in 1) post-operative states, 2) severe trauma, 3) severe burns, 4) sepsis & 5) postpartum
 - Factors: 1) dehydration, 2) GB stasis & sludging, 3) vascular compromise, 5) bacterial contamination 6) super infx

when you examine GB, you do NOT find gall stones or even upon imaging studies and exam

GB from outside, serosa is congested, hemorrhagic & inflamed
(acute cholecystitis)

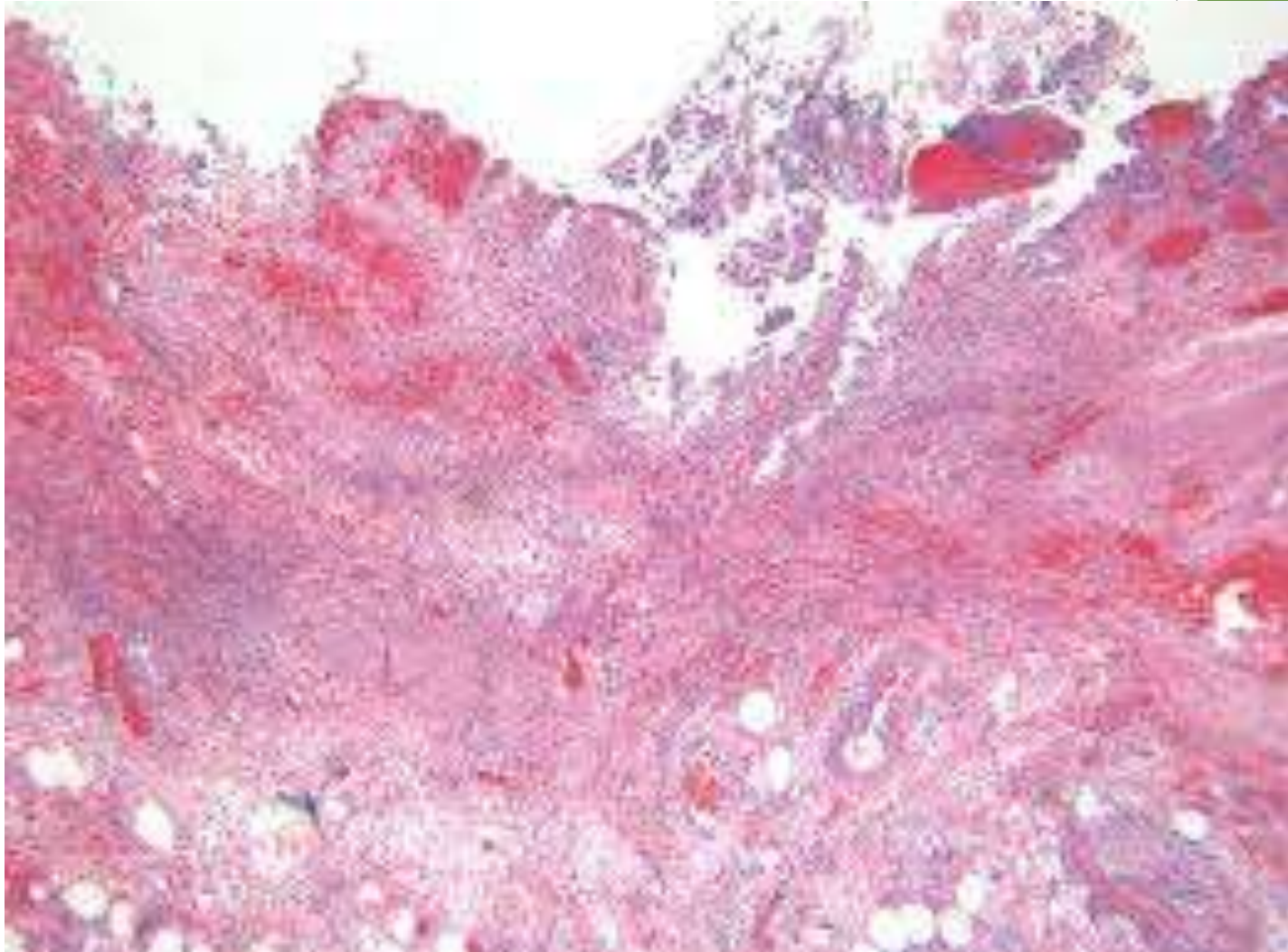


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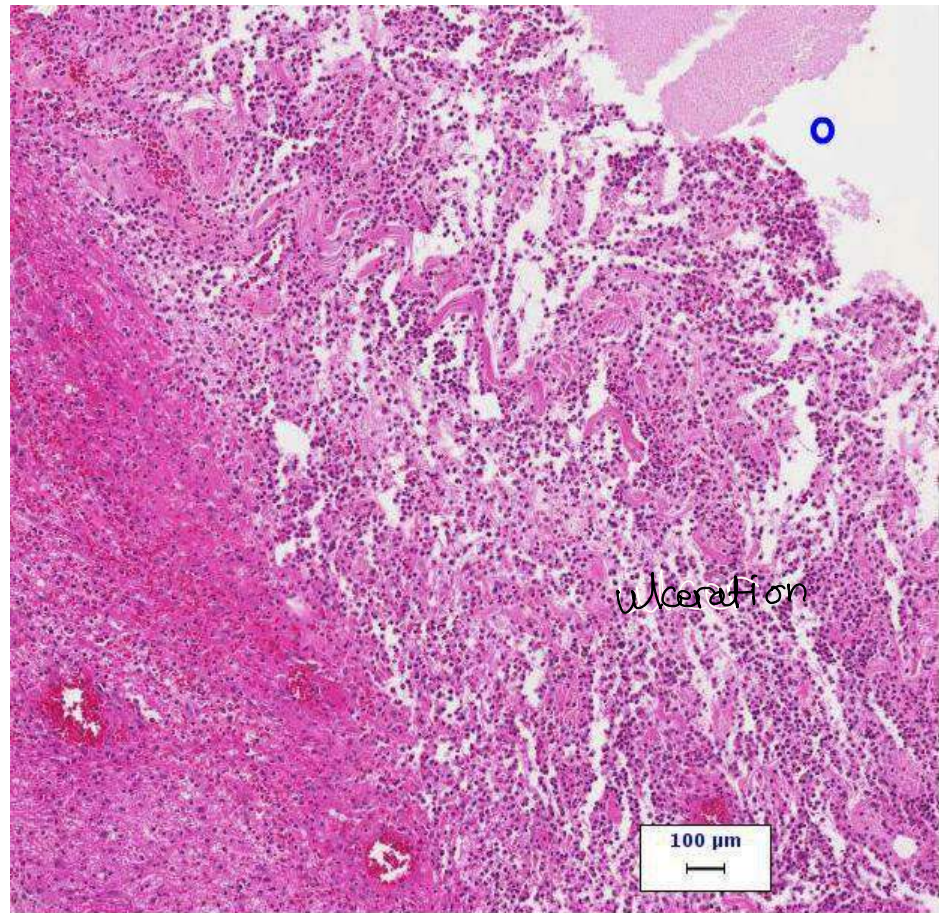
PATHOLOGY OF ACUTE CHOLECYSTITIS

- Enlarged (2-3x), tense GB with discolorations due to subserosal hemorrhages.
- Serosal fibrinous or suppurative exudate *formed of pus*
- Stones obstructing GB neck or cystic duct in 90%
- GB lumen filled with turbid bile, +/- fibrin, hemorrhage & pus
- Empyema of gallbladder: full of pus
- Thickened edematous hyperemic wall *(cardinal features of inflammation)*
- Gangrenous cholecystitis: black necrotic GB
- Hallmarks:* ➤ Histology: edema, WBC infiltration, congestion, abscess, hemorrhage & necrosis *↗*

mucosa is sluffed ^{removed} — ulcerated, hemorrhage, necrosis of the lining



acute inflammatory cells
infiltration ? blood & hemorrhage



ulceration

100 μm

100.00 μm

CHRONIC CHOLECYSTITIS

- ▶ +/- history of acute cholecystitis (NOT always)
- ▶ Gallstones almost always present,
- ▶ Supersaturation of bile predisposes to chronic inflammation & stone formation, NOT the obstruction. *it is related to the irritation & inflammation*
- ▶ Variable morphologic appearance: minimal changes, contraction, *start as flesh circles* enlargement, mucosal ulceration or wall thickening *Small* (X specific)
// // atrophy
- ▶ **Histology:** Mucosal ulcerations are infrequent; the submucosa and subserosa often are thickened from fibrosis, lymphocytes may be only clue of inflammation.

GB wall, thickened by fibrosis in chronic cholecystitis
// (// edema in acute)



pink → fibrosis & collagen deposition
which is the hallmark of chronic cholecystitis

flattened mucosa



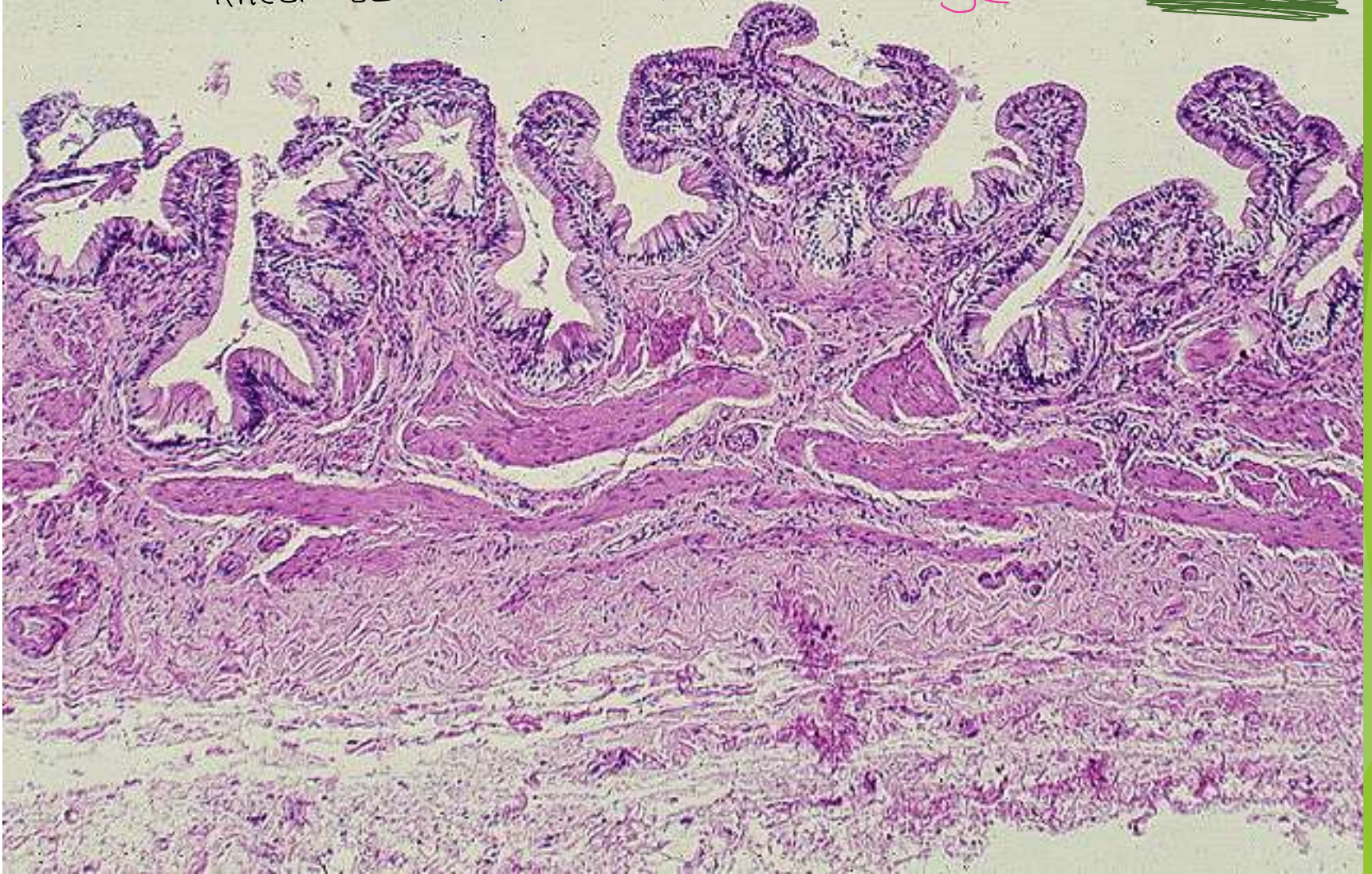
lining mucosa is preserved

no necrosis

no ulceration

no hemorrhage

fibrosis



CLINICAL FEATURES OF CHOLECYSTITIS

- ^{sudden onset} Acute & ^{more gradual insidious onset} chronic calculous cholecystitis have similar & variable symptoms: minimal nonspecific symptoms to biliary colics to severe **RUQ pain** ^{typical presentation}
- Fever, nausea, ^{↑ WBC} leukocytosis. ^{↑ vomit} ^{in peripheral blood}
- Acute acalculous cholecystitis: symptoms obscured by general condition ^{? dehydrated, bed ridden}
- **Dx: Ultrasonography**
- ^{with late dx → ascending to bile ducts} **Complications:** cholangitis, sepsis, GB perforation, abscess, rupture, cholecyst-enteric fistula, intestinal ileus, ... ^{between GB & small bowel}

TUMORS OF THE GALLBLADDER

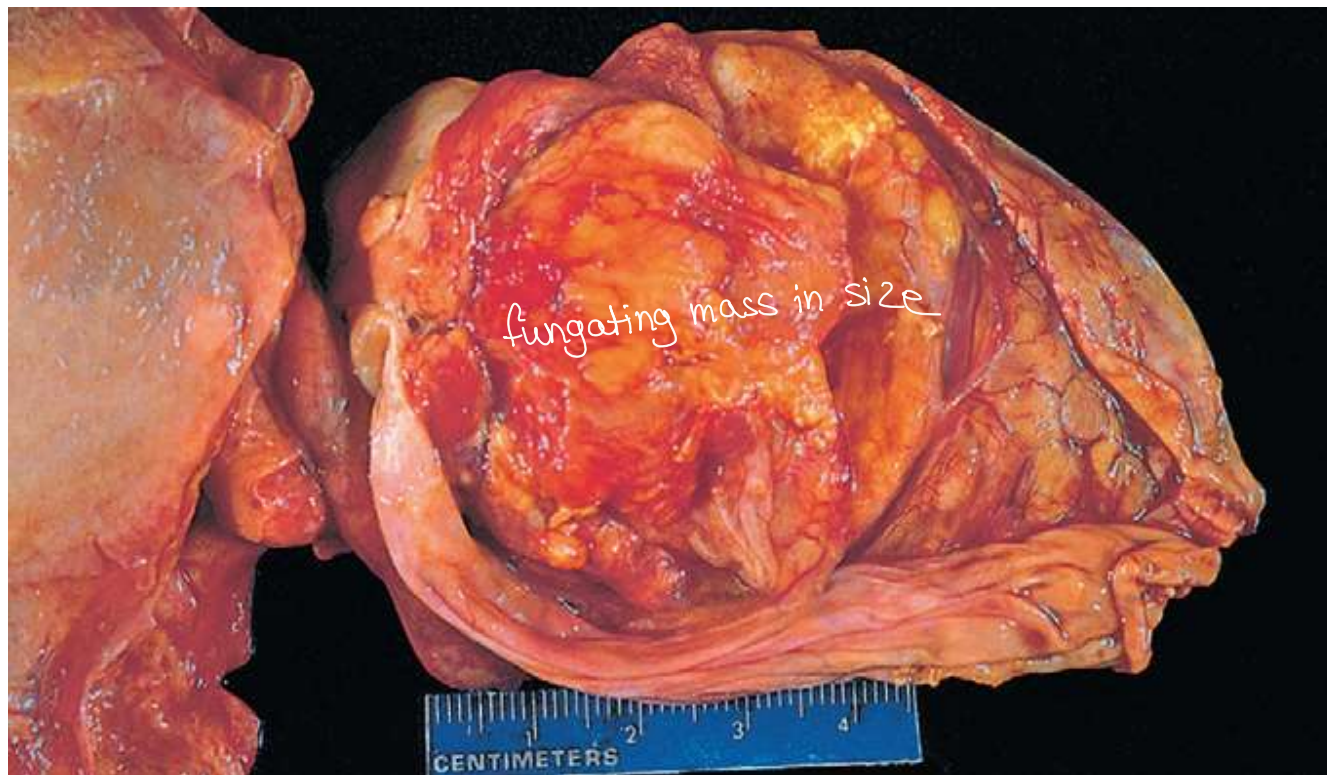
GALLBLADDER CARCINOMA

- Commonest extrahepatic biliary tract cancer
- More common in women; peak 7th decade
- Due to recurrent trauma and inflammation: usually associated with stones;
- Morphology: Infiltrating or fungating growth pattern *protruded to the lumen of GB*
- Most are adenocarcinoma.

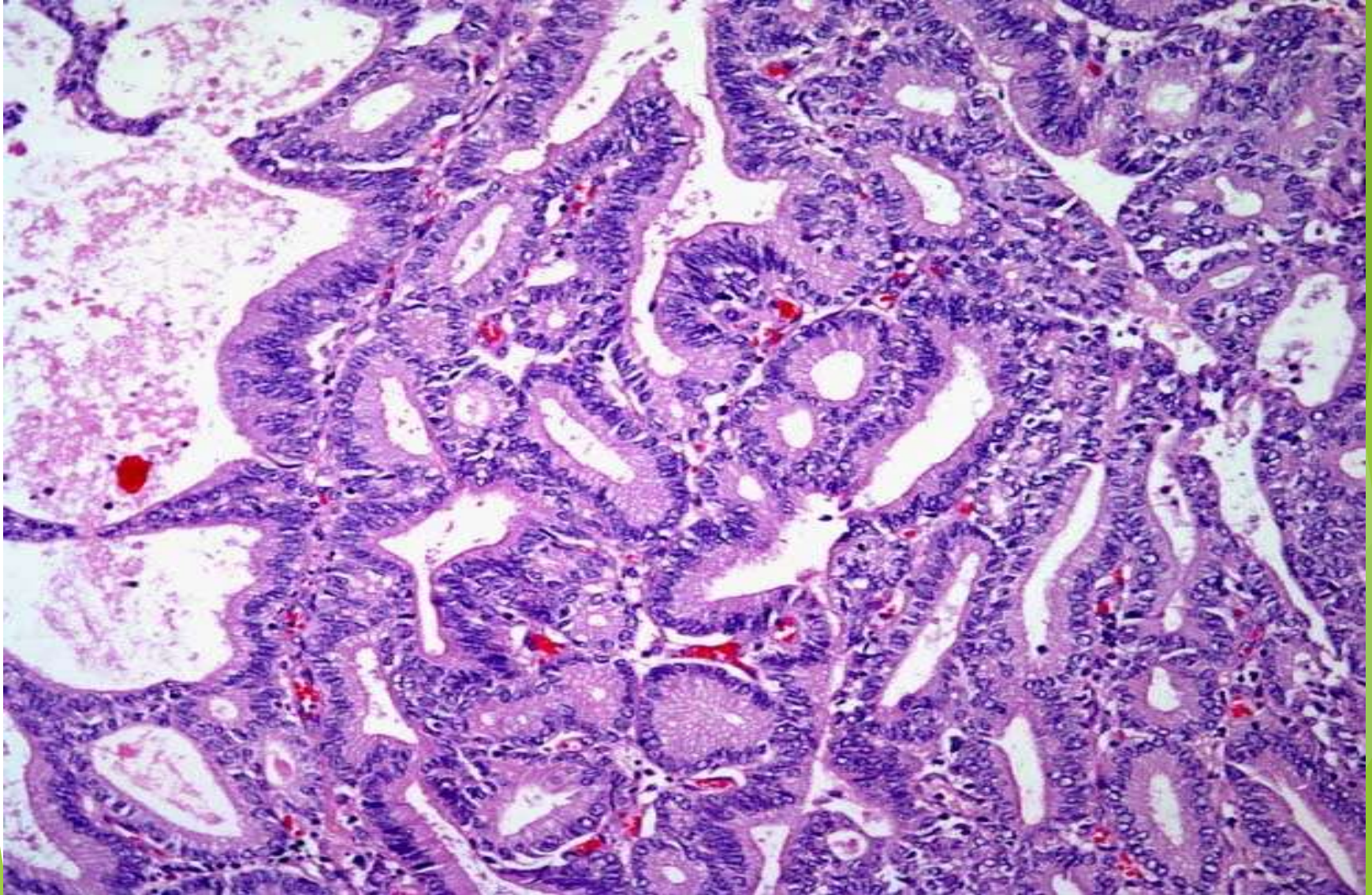
- *(gradual)* Insidious symptoms similar to cholelithiasis *or chronic cholecystitis → masking of symptoms & late dx*
- If obstruction develops early: early diagnosis and treatment. *→ with more survival rate*
- Advance stage at diagnosis (late) *obscuring of symptoms by other diseases or ? can be asymptomatic*
- *late stage* Seeding to peritoneum, GIT, and lungs, *met + lungs mets*
- **Prognosis: dismal, 5 year survival: 1%**

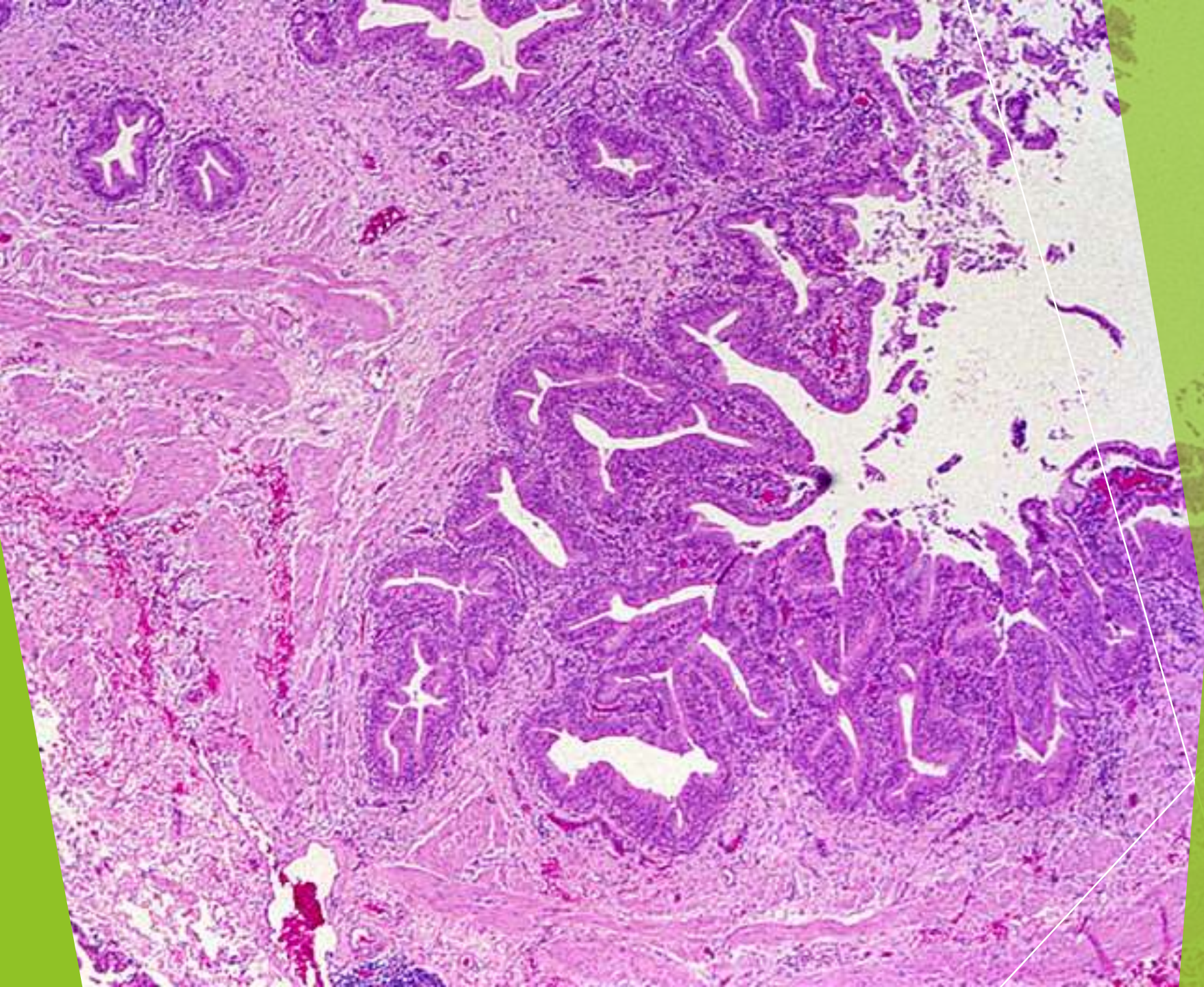
dx → mostly incidental finding during examination of GB removed for acute or chronic cholecystitis

Bad tumor, discovered late



adenocarcinoma
usually infiltrates walls & muscle wall of GB





A histological slide stained with hematoxylin and eosin (H&E) showing a dense population of tumor cells. The cells are arranged in nests and cords, with some showing glandular formation. They are infiltrating through a pink-stained muscle layer and surrounding nerve bundles, which are visible as pale, fibrous structures. The overall architecture is disrupted by the invasive growth of the tumor cells.

infiltrate or surround muscle layer or nerves

infiltrating adenocarcinoma of GB with the surrounding inflammatory reaction

