GBS, pathogenesis and complications

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Types of gallstones

- Mixed:
- cholesterol content 50-80%.
- various shape and colors
- usually small multiple stones of faceted surface.

- Pure cholesterol:
- cholesterol content around 100%.
- pale yellow
- usually large solitary.

Types of gallstones

- Pigmented: cholesterol content less than 20%.

- Black stones :
- Pts with cirrhosis and hemolysis.
- Homogenous, brittle
- Usually small multiple stones.
- Present in the GB

- Brown stones:
- After biliary tract infection.
- Usually small multiple stones
- Present in the biliary tree

Risk factors

- “Female, Fat, Forty, Fertile”
- Oral contraceptives
- Obesity
- Rapid weight loss
- Fatty diet
- DM
- Prolonged fasting
- TPN
- Ileal resection
- Hemolytic states
- Cirrhosis
- Bile duct stasis (biliary stricture, congenital cysts, pancreatitis, sclerosing cholangitis)
- IBD
- Vagotomy
- Hyperlipidemia

Pathogenesis of gallstones

- **<u>Cholesterol GS</u>**:
- Bilirubin, bile salts, phospholipids, and cholesterol are the major organic solutes in the bile.

- Cholesterol is insoluble in water, and thus in bile.

- Cholesterol is secreted from the liver with carrier (lecithin). This union called vesicles.

- Bile salts dissolve these vesicles to form soluble aggregate called mixed micelles (occurs mainly in the GB)

Pathogenesis of gallstones

- The main substances in GS formation are cholesterol and Ca bilirubinate
- GS formation occurs when these substances approach the limits of their solubility.
- Concentration of bile in the GB lead to more super saturation with these substances, which will precipitate as a microscopic crystals.
- These crystals are trapped with GB mucus producing GB sludge (mucus gel), worsening leads to crystal formation.
- Over time the crystals grow, aggregate to form macroscopic stones

Pathogenesis of gallstones

- So, the main factors for cholesterol GS formation are:

  - imbalance between cholesterol and carriers
  - degree of bile concentration and stasis in the GB

Pathogenesis of gallstones

- **<u>Pigmented GS</u>:**

- <u>Black stones:</u>
  - over production of indirect bilirubin (hemolysis, cirrhosis)

- Usually are not associated with infected bile.

- Located almost exclusively in the GB.

- main component is Ca bilirubinate

Pathogenesis of gallstones

- <u>Brown stones:</u>
  - Typically found in the bile duct as a primary stones.

  - Contain more calcium palmitate.

  - Usually secondary to bacterial infection (bacterial enzyme is phospholipases, hydrolyzes the lecithin to form palmitate)

- **<u>Stages</u>**:

  - Lithogenic state (GS formation), stages
  - Asymptomatic GS
  - Symptomatic GS (biliary pain)
  - Complicated cholelithiasis:

    * Acute cholecystitis with it is complications.
    * Chronic cholecystitis.
    * Choledocholithiasis with and without cholangitis.
    * Biliary pancreatitis.
    * Gallstone ileus.
    * Gallblader carcinoma.

Acute cholecystitis

- Acute inflammation of the gallbladder induced by obstruction secondary to GS or sludge in 90-95% of cases.

- Following this early phase, 20-50% of patients manifest a proliferation of bacteria, resulting in secondary bacterial infection of the organ.

- most cases are mild to moderate in severity, 5-10% have progression into complications (emphysematous cholecystitis, empyema, and perforation).

Acute cholecystitis

- Types:
- Calculous (90%-95%).

- Acalculus (5%-10%):
- Stasis and ischemia.
- Critically ill patients.
- High mortalitiy (40%).

- Incidence:
- 70%-80% patients remain asymptomatic through life.
- 25% of women >60 yr old have GBS, 10% will have symptoms after 5 yrs and 20% after 20 yrs
- Risk of symptoms is 1%-3%/yr.
- Acute cholecystitis usually occurs in symptomatic group.

Acute cholecystitis

- Xanthogranulomatous cholecystitis:
- Leakage of bile into the wall
- Inflammatory reaction with formation of xanthoma cells
- More acute presentation and more complications
- US diagnosis is rare, presence of intramural nodules overlap with GB Ca.

- Emphysematous cholecystitis:
- Gas forming bacteria
- Early complications
- High morality 15%-25%
- 1% of all cases of acute cholecystitis
- More in diabetic male pts

Acute cholecystitis

- Clinical features:

- Pathogenesis:

- Complications:
- Empyema, perforation, fistula.

- Diagnosis:
- Plain X ray
- US
- CT scan
- ERCP,MRCP
- HIDA scan

- Management:

Chronic cholecystitis

- may arise from repeated attacks of symptomatic acute cholecystitis or it develops without any history of acute attacks.
- almost always associated with gallstones.
- Macroscopic findings: gallbladder may be contracted (from fibrosis).

biliary dyskinesia

- Cholecystokinin-Tc-HIDA scan (ejection fraction of <35% at 20 min of cholecystokinin is diagnostic).
- Most pts have evidence of chronic chole.
- > 50% of pts have improvement after cholecystectomy, so it is indicated.

Biliary pain

- Biliary colic is a misnomer
- Pain is visceral poorly localized pain
- No guarding or rebound tenderness
- No fever
- Duration of pain usually 1-5 h, beyond 24h suggest of cholecystitis
- Time of pain.

Choledocholithiasis

- Incidence:
- Types:
- Secondary:
- Primary:

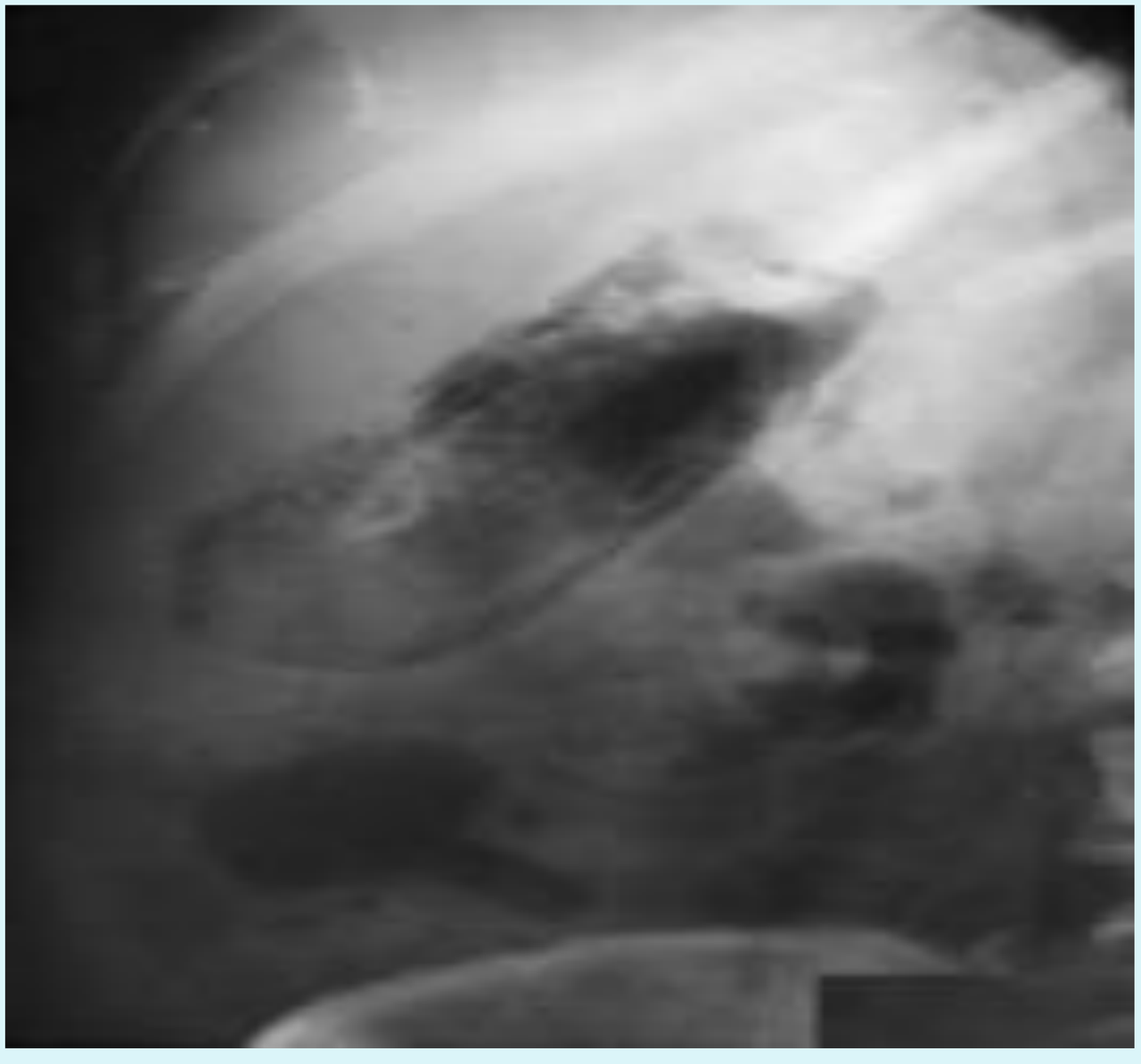
- Clinical picture and complications.
- Diagnosis (clinical, biochemical, and radiological)
- Management.

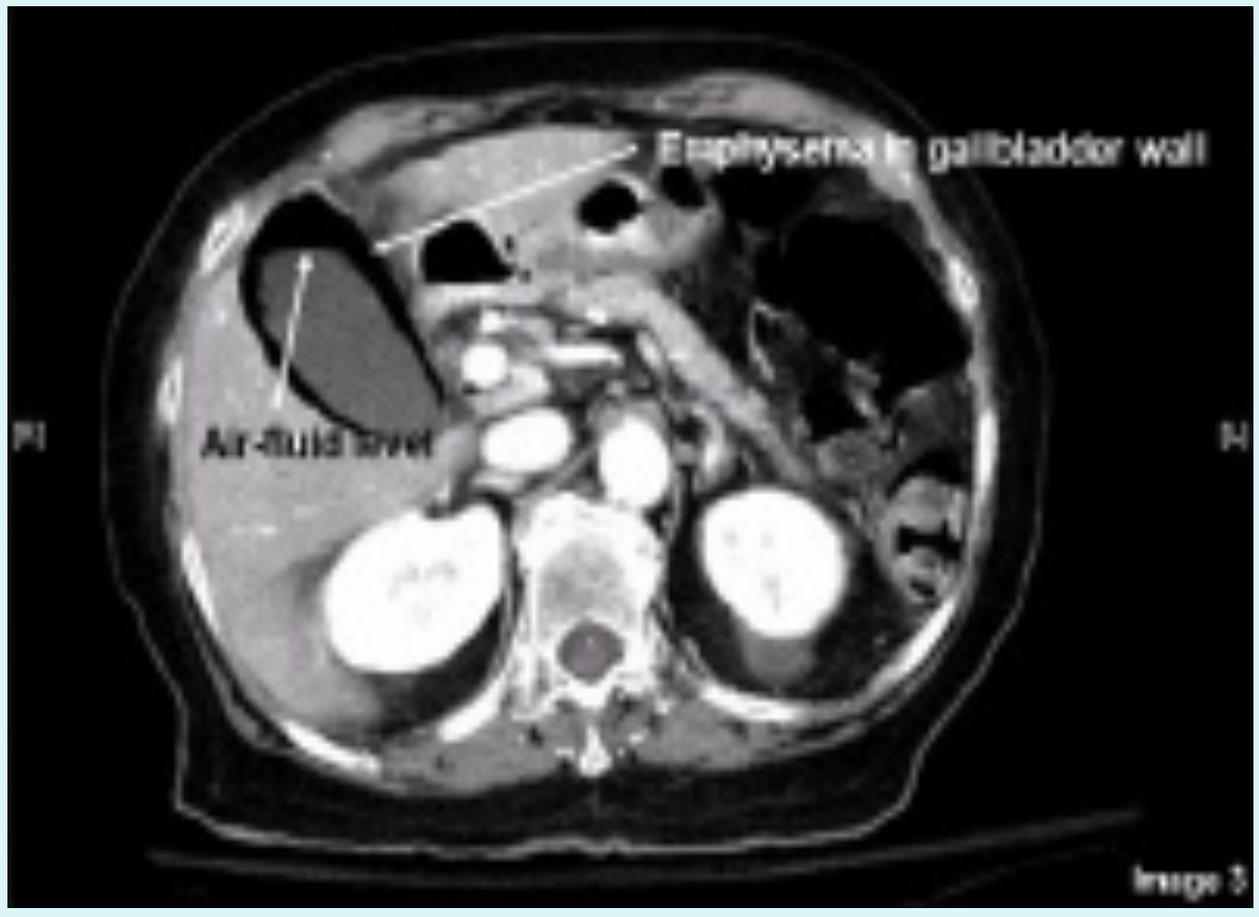
Biliary pancreatitis

- Among patients with gallstones, 4-8% will present acute pancreatitis.
- In patients with multiple calculi less than 3 mm in diameter (microlithiasis) the risk is up to 30%.
- Gallstones are responsible for 50% of all cases of pancreatitis.
- 90% have mild to moderate self limited pancreatitis.

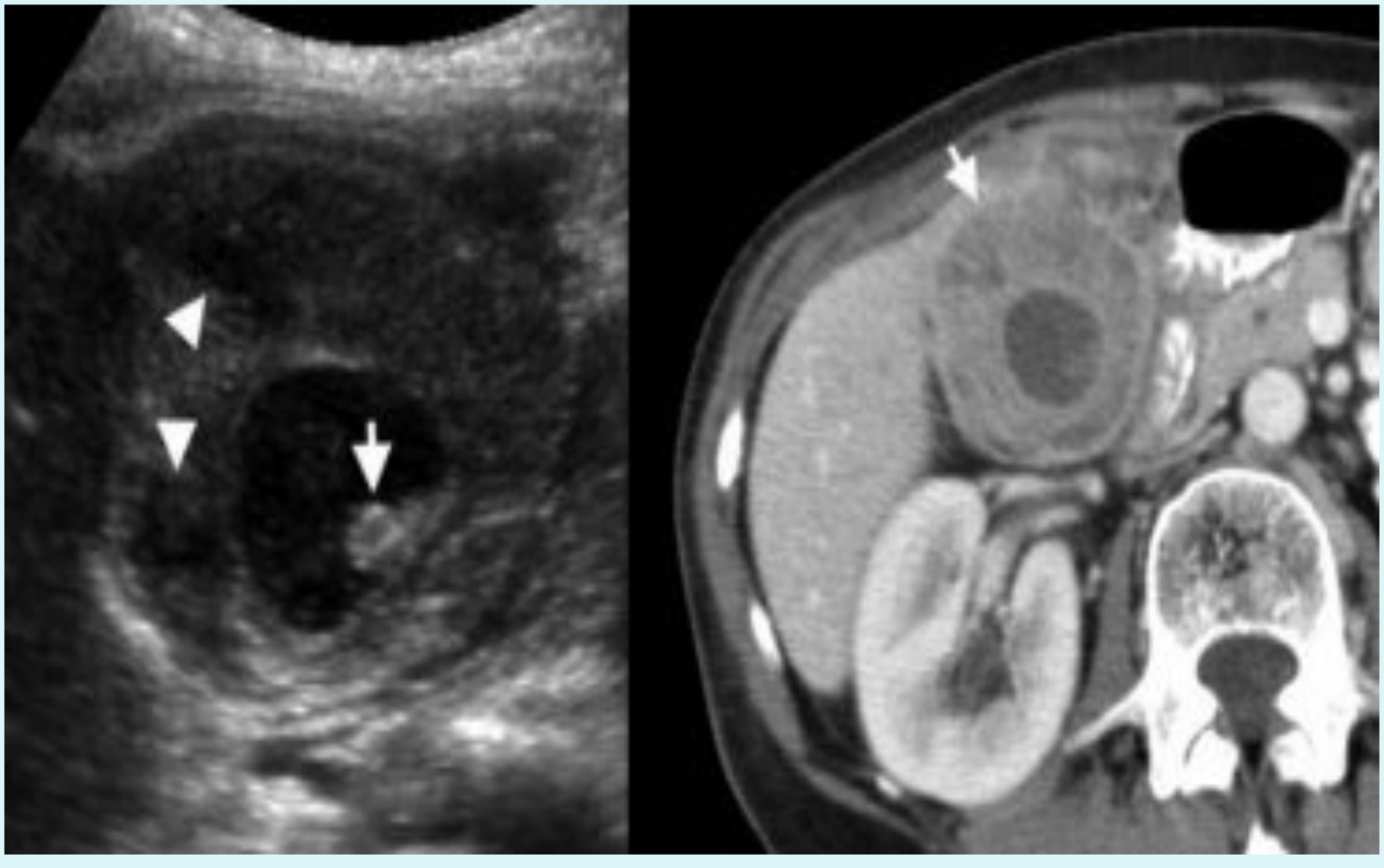
Indications for Prophylactic Cholecystectomy

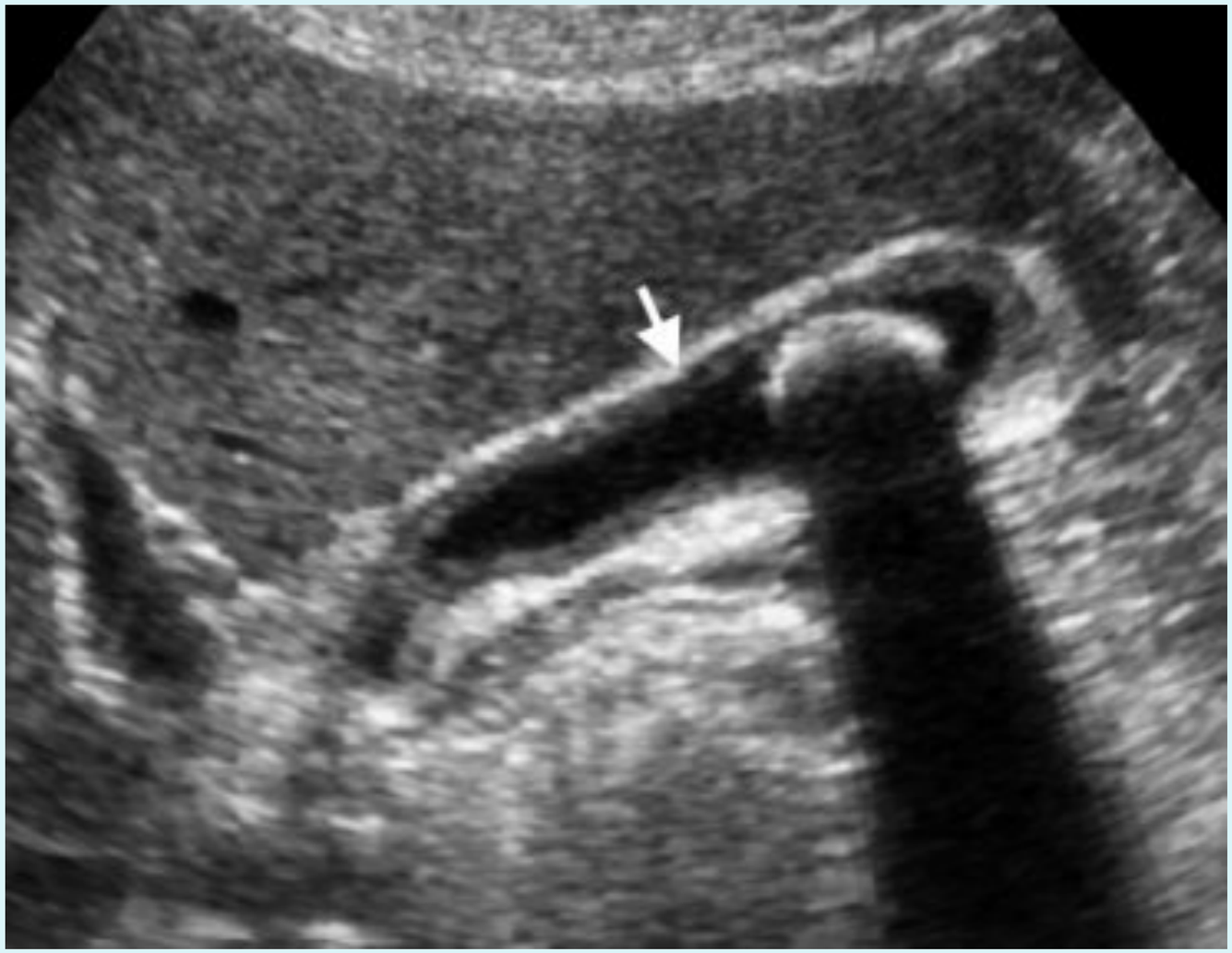
- Pediatric gallstones.
- Congenital hemolytic anemia.
- Gallstones >2.5cm.
- Porcelain gallbladder (25% risk of Ca).
- Incidental gallstones found during intraabdominal surgery.
- Recommended prior to transplantation.
- in young women.
- GB polyp > 1 cm (risk of malgnancy)

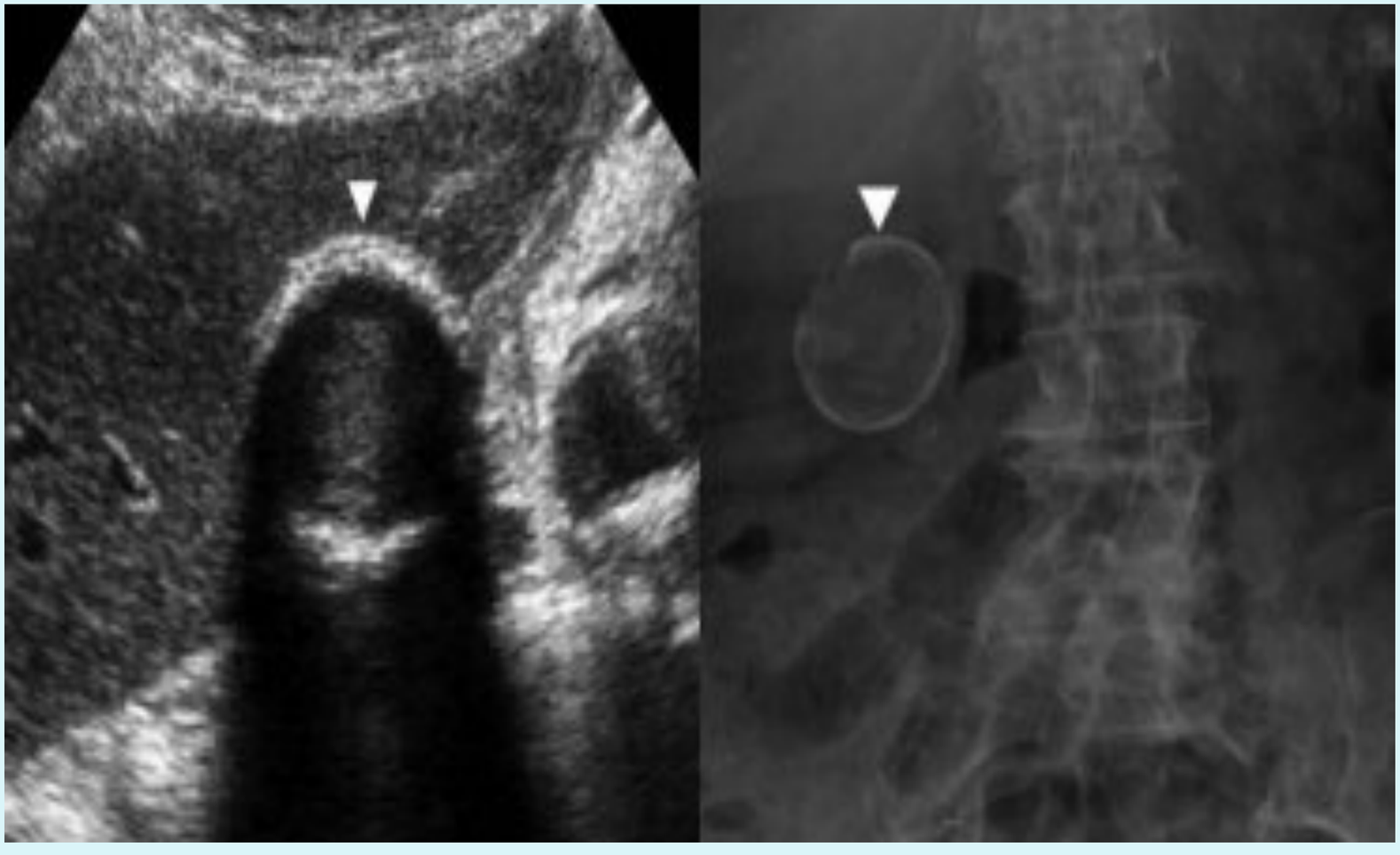



Emphysema in gallbladder wall
Air-fluid level
Image 3

Xanthogranulomatous cholecystitis







Thank you