The Hepatobiliary Acute Abdomen

What does your clinician want to know?

Presented by Dr Nethmee Malla

BMUS General Medicine Study Day

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Dr Dean Huang Prof Paul Sidhu

Dr Maria Sellars Dr Anamaria Deganello

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Objectives

1. **Map the causes**
   Hepatobiliary causes of the acute abdomen

2. **Correlate the picture**
   Match the clinical & biochemical picture to ultrasound findings

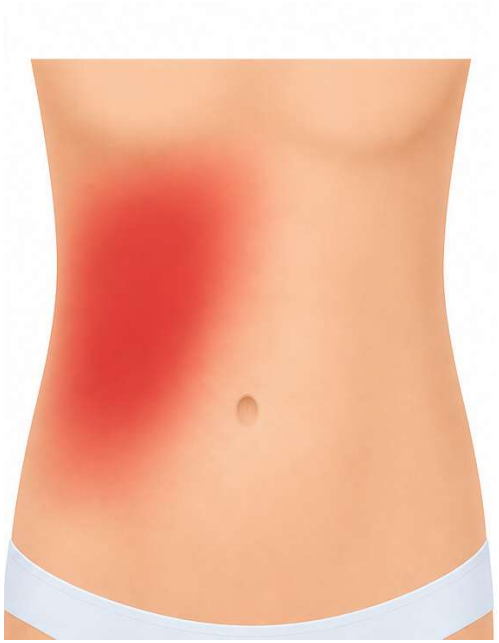
3. **Know the pathologies**
   Common conditions and their ultrasound (and cross-sectional) findings

4. **Define ultrasound’s role**
   When is it useful vs less useful — and what does your clinician want to know?

PART 1

Hepatobiliary Causes of the Acute Abdomen

(and in particular, RUQ pain)



LIVER PARENCHYMA

The functional tissue of the liver responsible for metabolism, detoxification and bile production.

GALLBLADDER

Stores and concentrates bile produced by the liver and releases it into the biliary tree.

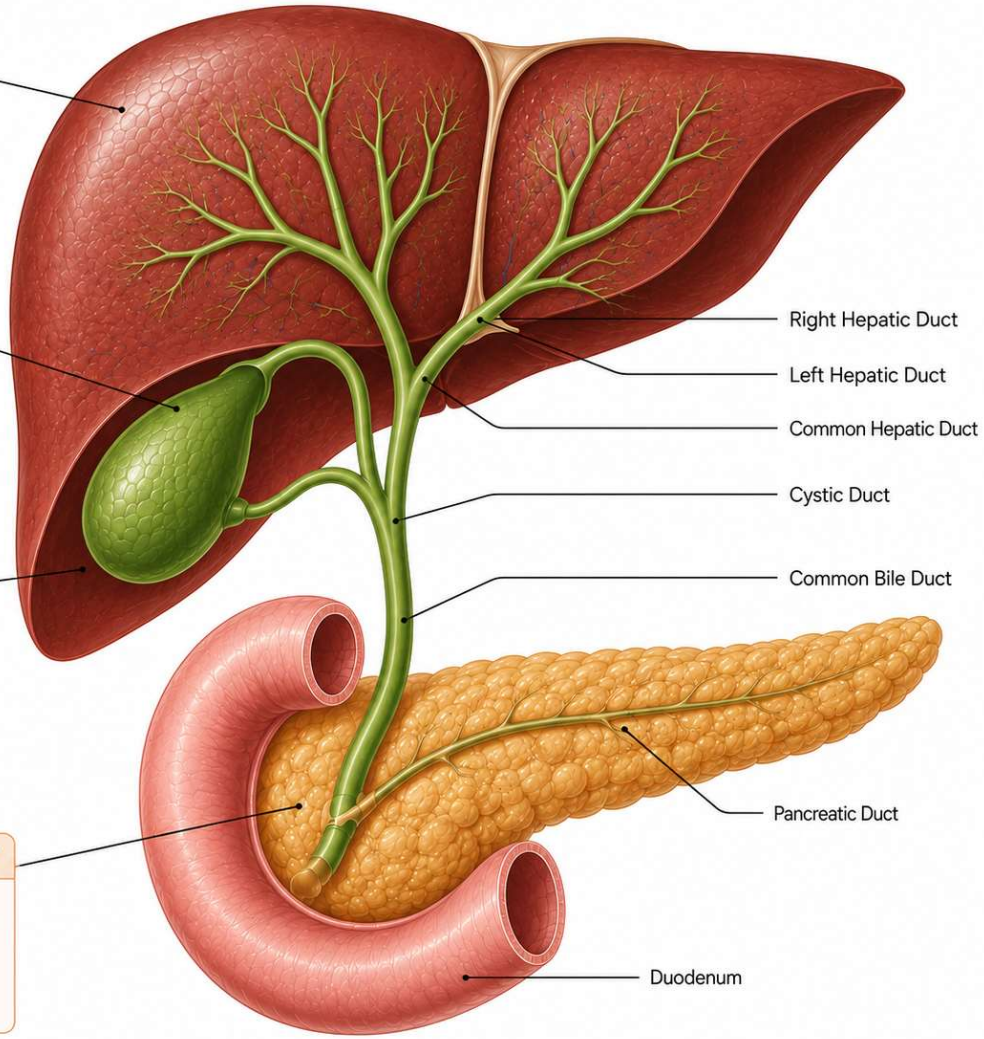
BILIARY TREE

Network of ducts that transports bile from the liver to the duodenum.

- Right & Left Hepatic Ducts
- Common Hepatic Duct
- Cystic Duct
- Common Bile Duct

PANCREATIC PATHOLOGY

The pancreas lies adjacent to the biliary tree. Pathology such as pancreatitis, masses or cysts can affect the pancreas and biliary drainage.



Hepatobiliary causes of the Acute Abdomen

Gallbladder Disease

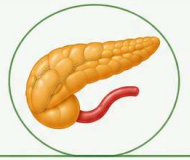
- Cholecystitis
- Biliary colic
- Empyema
- Gangrene
- Perforation

Liver Conditions

- Hepatitis
- Abscess
- Congestion
- Budd–Chiari
- Malignancy (primary, metastases)

Bile Duct Disorders

- Choledocholithiasis
- Ascending Cholangitis
- Mirizzi Syndrome
- Stricture
- Malignancy



Pancreas

- Acute Pancreatitis (gallstone or alcohol)
- Pseudocyst

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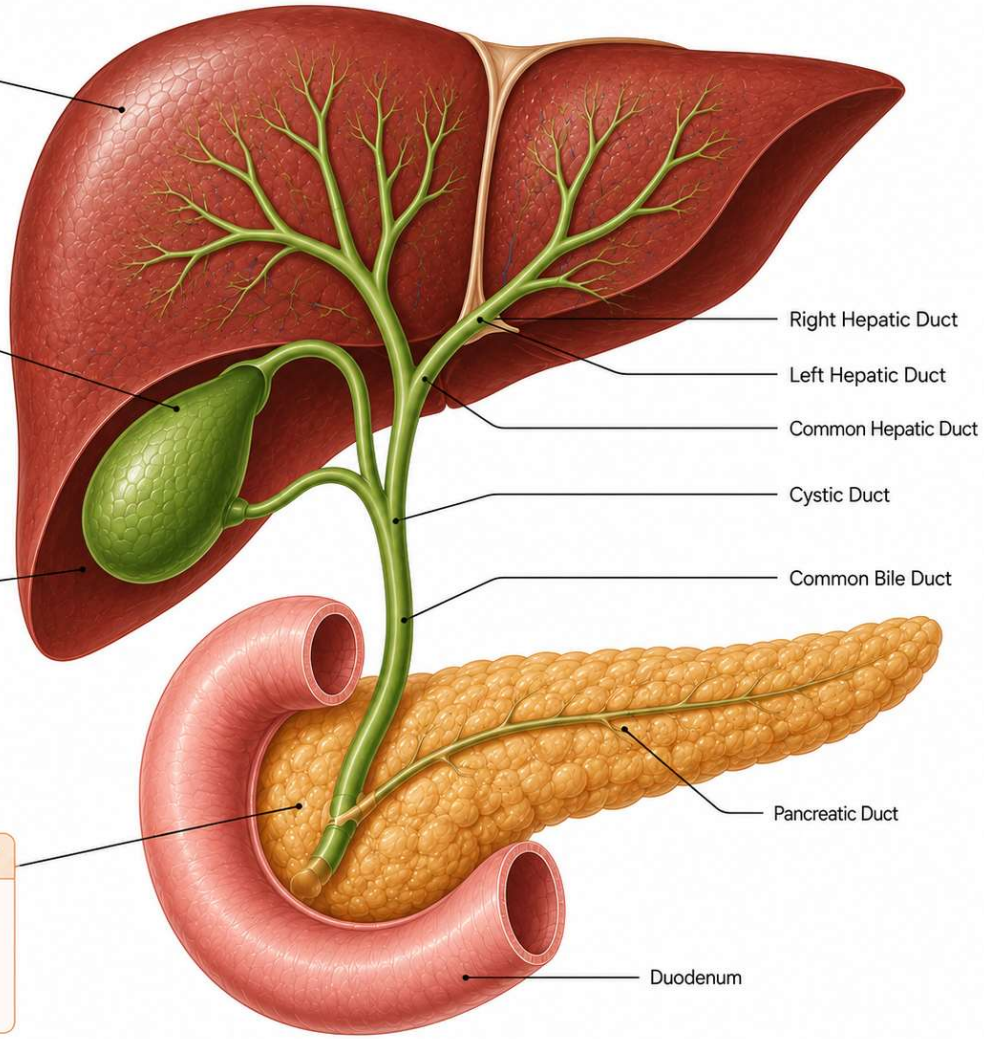
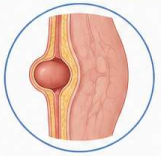


OTHER CAUSES / MIMICS

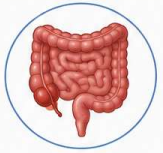
Kidney Stones
Renal colic

Basal Pneumonia
Right lower lobe infection

Peptic Ulcer
Perforation or penetration



Hernia
Abdominal wall herniation



Appendicitis
Retrocecal appendix irritation

AAA
Abdominal aortic aneurysm

PART 2

Clinical Presentation

Clinical Presentations

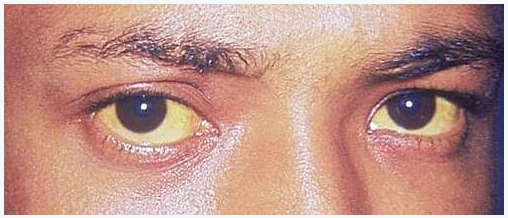
Biliary colic
Post-prandial, episodic RUQ or epigastric pain settling within hours; bloods often normal.

Acute cholecystitis
Constant RUQ pain >6 h, fever, positive Murphy's sign; ↑WCC & CRP, LFTs mildly deranged.

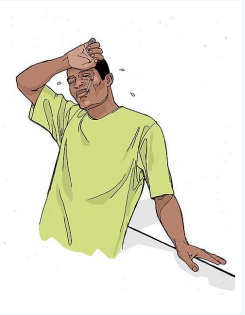
Choledocholithiasis
Stones – often obstructing – in CBD.

Ascending cholangitis
Charcot's triad: pain, jaundice and fever with rigors.
Reynolds' pentad: + hypotension and confusion (suppurative, septic).

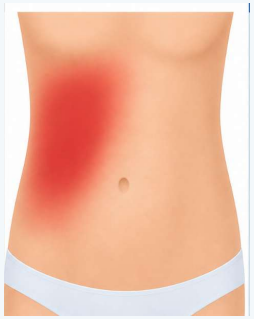
Red flags
Peritonism or instability → urgent CT and surgical review.



Jaundice



Fever (often of unknown origin)



Right upper quadrant pain

Abnormal LFTs

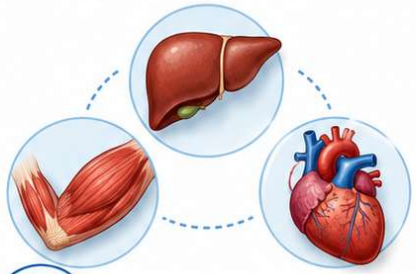
Abnormal LFTs: types of liver enzymes

ALT
Alanine Aminotransferase

Liver Cell Injury

Most specific for liver damage

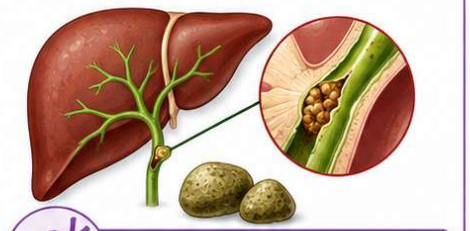
AST
Aspartate Aminotransferase



Liver, Muscle, Heart

Less specific for liver damage

ALP
Alkaline Phosphatase



Bile Duct Obstruction

Cholestasis / Gallstones

GGT
Gamma-Glutamyl Transferase

Bile Duct Issues

Alcohol & Bile Disease

ALT
AST

Hepatocytes hold ALT & AST — they spill into blood when liver cells are injured (hepatocellular).

ALP
GGT

Bile ducts line up ALP & GGT — they climb with cholestasis/obstruction as bilirubin backs up (cholestatic).

Bilirubin

Bilirubin backs up when bile can't drain: conjugated (direct) signals obstruction; unconjugated (indirect) points to haemolysis.

The pattern tells you which compartment is hit.

Lab Interpretation

↑ High ALT & AST	→	Liver Cell Injury
↑ High ALP & GGT	→	Bile Duct Obstruction
AST > ALT	→	Alcohol Related Damage

Abnormal LFTs: patterns

Marker	Hepatocellular	Cholestatic / Obstructive
ALT / AST	↑↑↑ (often >5× ULN)	↑ mild
ALP	↑ mild	↑↑↑
GGT	↑ mild	↑↑ confirms biliary origin
Bilirubin	variable	↑ conjugated
Typical causes	hepatitis, toxins, ischaemia	stones, stricture, tumour

Obstructive vs Non-obstructive Jaundice: Interpreting Bilirubin

PRE-HEPATIC
Haemolytic

↑ Unconjugated (indirect) bilirubin

- Haemolysis, Gilbert's, sepsis
- Not a biliary problem
- ALP & transaminases normal

HEPATIC
Hepatocellular

Mixed bilirubin

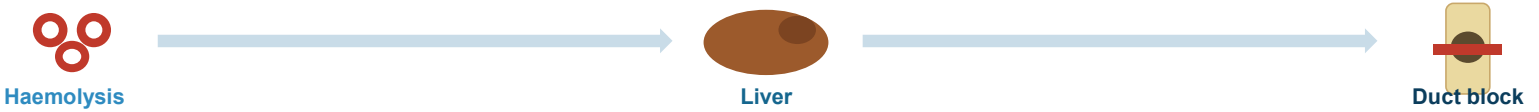
- Hepatitis or cirrhosis
- ↑ ALT & AST, well above ALP
- Cholestatic picture can overlap

POST-HEPATIC
Obstructive

↑ Conjugated (direct) bilirubin

- Stone, stricture or tumour
- ↑ ALP & GGT
- Pale stool, dark urine, pruritus

BILIRUBIN PATHWAY
where the block sits



Timing
Bilirubin can rise late — a normal value does not exclude early obstruction. ALP & GGT often climb first; rising ALP and bilirubin together signal decompensating obstruction.

Courvoisier's law
Painless jaundice with a palpable gallbladder means malignancy until proven otherwise.

Ultrasound
Is the biliary tree dilated? That single answer separates obstructive from non-obstructive jaundice, and is the main role of ultrasound here.

Acute Pancreatitis

Pancreatic & inflammatory markers

PANCREATIC MARKERS

What the enzymes ask of the scan

Amylase & lipase

>3× ULN confirms acute pancreatitis; lipase is more specific and stays raised longer in late presenters.

Gallstones first

Commonest UK cause — your early US targets the source: gallbladder stones, CBD calibre and duct dilatation.

Severity & CT timing

Enzymes don't grade severity — contrast CT at 72 h+ shows necrosis; early US is for cause, not staging.

INFLAMMATORY MARKERS

Signals that change the scan

CRP at 48 h

>150 mg/L flags severe or necrotising disease — the trigger to plan contrast-enhanced CT.

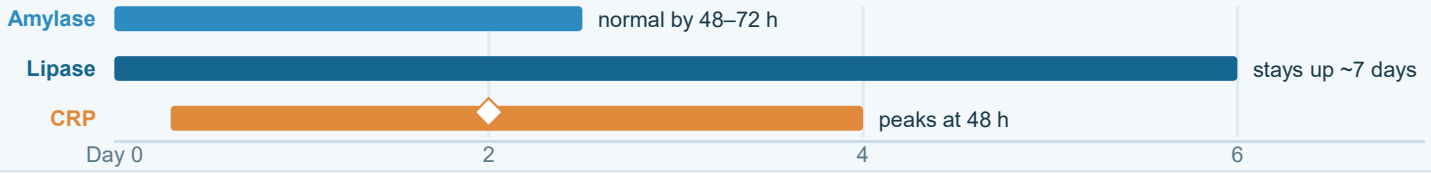
WCC & CRP

Confirm inflammation but never localise it; correlate with the clinical picture and the scan.

Rising lactate

Perfusion & severity marker. **Differential:** jaundice + rising lactate suggests cholangitis — fast-track US.

MARKER TIME COURSE
rises fast vs lingers



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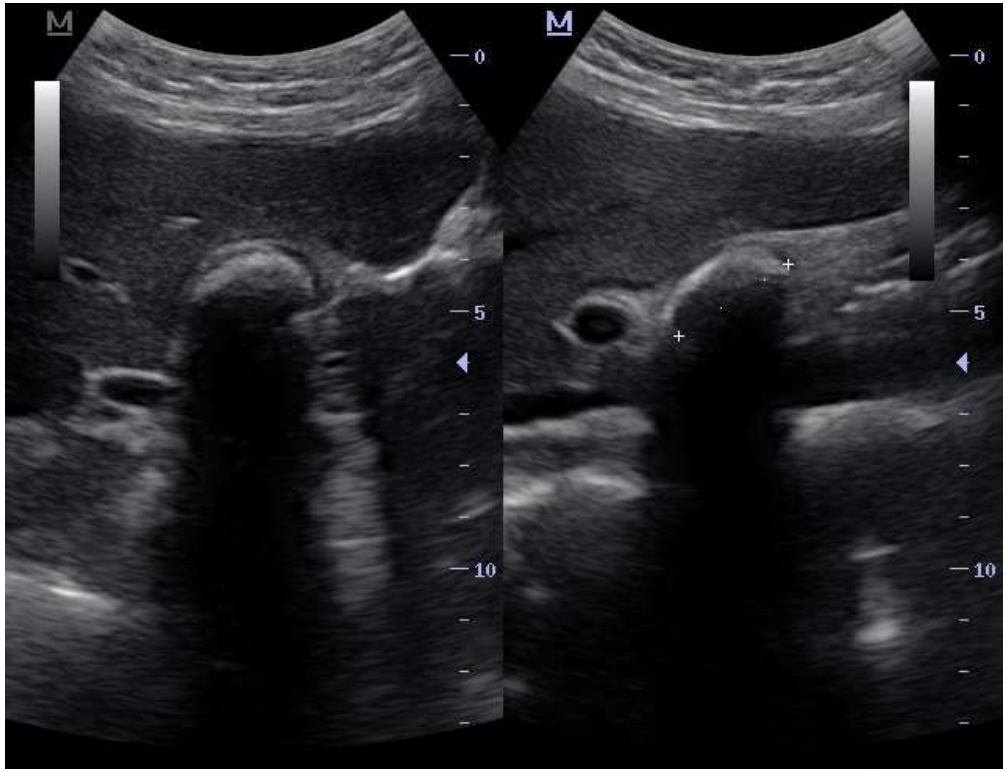
PART 3

Common acute pathologies

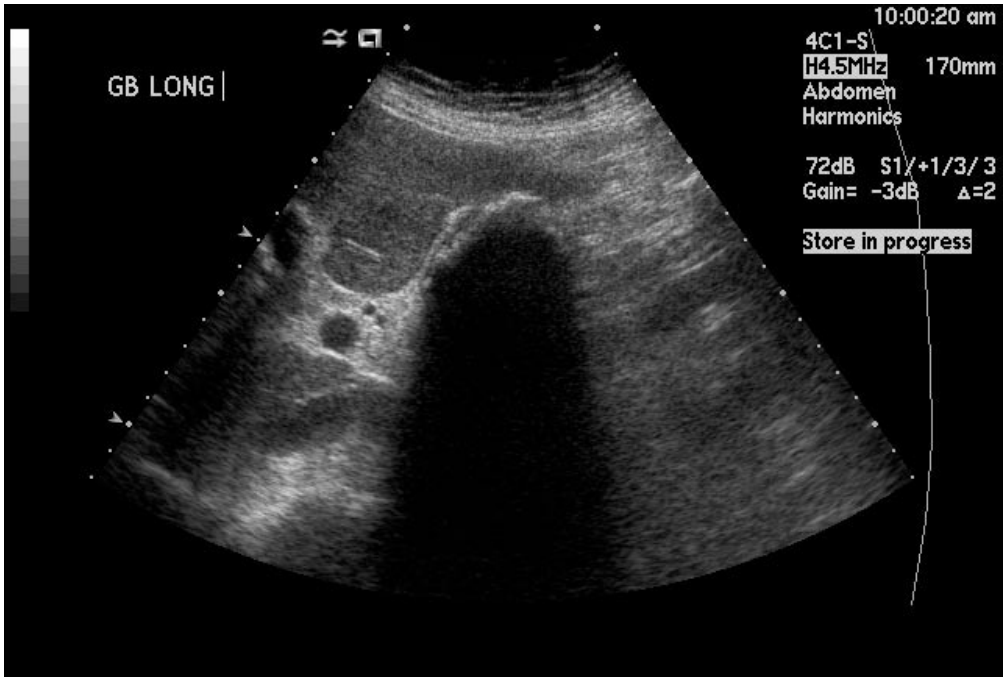
Gallstone disease

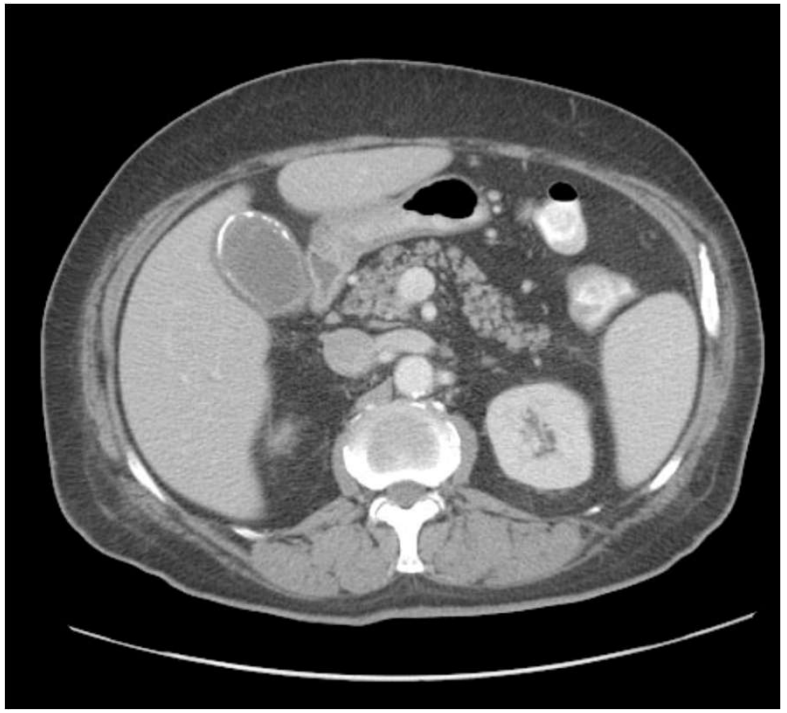
- **US is the gold standard:** over 95% sensitivity for gallstones
- **Scan fasted:** a contracted, post-prandial gallbladder hides stones (fast 6 h)
- **Classic triad:** echogenic focus, posterior acoustic shadowing, mobility with position
- **Small stones or sludge:** shadowing may be absent, so roll the patient to confirm mobility
- **WES sign:** wall, echo and shadow when the gallbladder is packed with stones
- **Always report:** stone presence, size of the largest, wall thickness, CBD calibre



Wall-Echo Sign (impacted gallbladder)

Gallstone disease: porcelain gallbladder





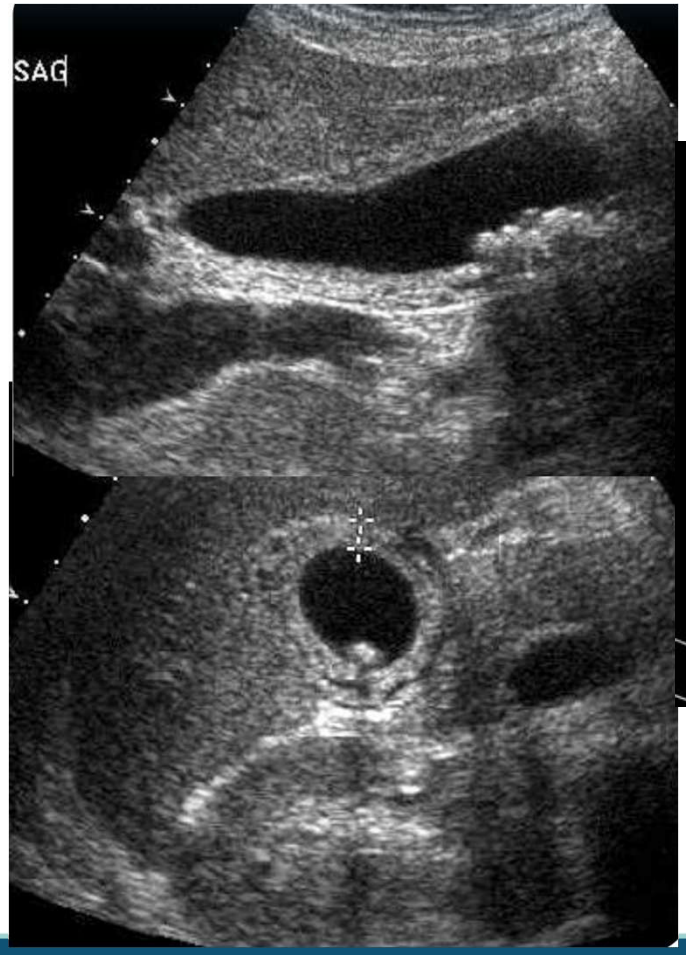
Porcelain Gallbladder

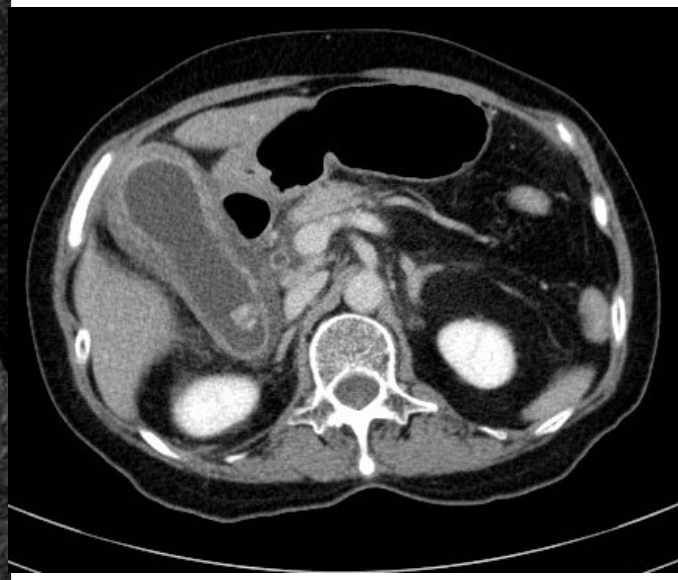
Gallstone disease: acute cholecystitis

- Acute vs chronic
- **Sonographic Murphy's sign:** maximal tenderness over the gallbladder under the probe
- **Wall thickening:** over 3 mm with a matching clinical picture

What your clinician wants to know

- ✓ Presence or absence of gallstones
- ✓ GB wall thickening: striated? Gas?
- ✓ Biliary dilatation
- ✓ Pericholecystic fluid, generalized free fluid
- ✓ Sonographic Murphy's positivity
- ✓ PV/HA patency





Gallstone disease: emphysematous cholecystitis

- Associated with acalculous cholecystitis
- Associated with vascular compromise of the cystic artery
- Usually very unwell

What your clinician wants to know

- ✓ Presence or absence of gallstones
- ✓ GB wall thickening: striated? Gas?
- ✓ Biliary dilatation
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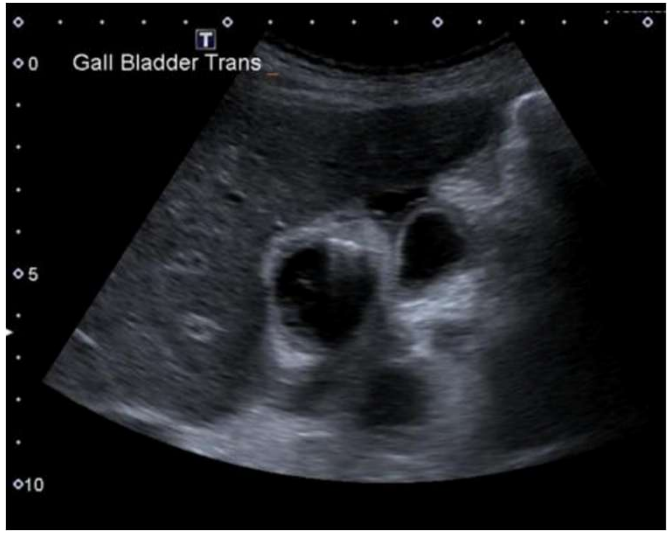
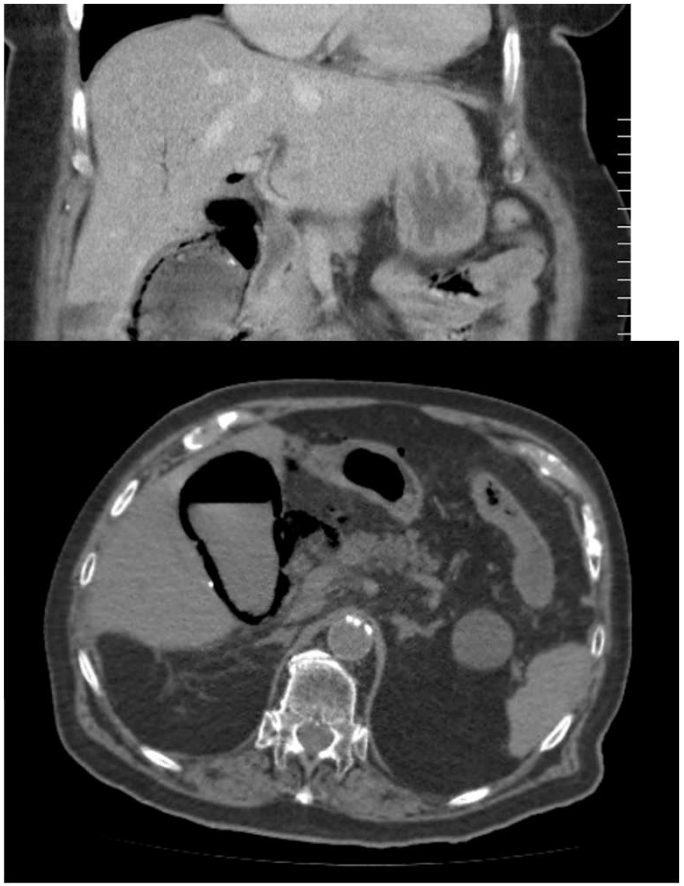


Acute Emphysematous Cholecystitis



Choledocholithiasis (CBD stones)

- **Normal CBD:** up to 6-7 mm, plus about 1 mm per decade over 60, and up to 10 mm after cholecystectomy
- **Distal CBD:** often gas-obscured, so a non-dilated duct does not exclude a stone

- **US has limited sensitivity for CBD stones -** as stones hide in the distal duct behind gas
- **Clinical presentations:** ascending cholangitis, acute pancreatitis, biliary colic
- Approx. 20% of CBD stones will not shadow

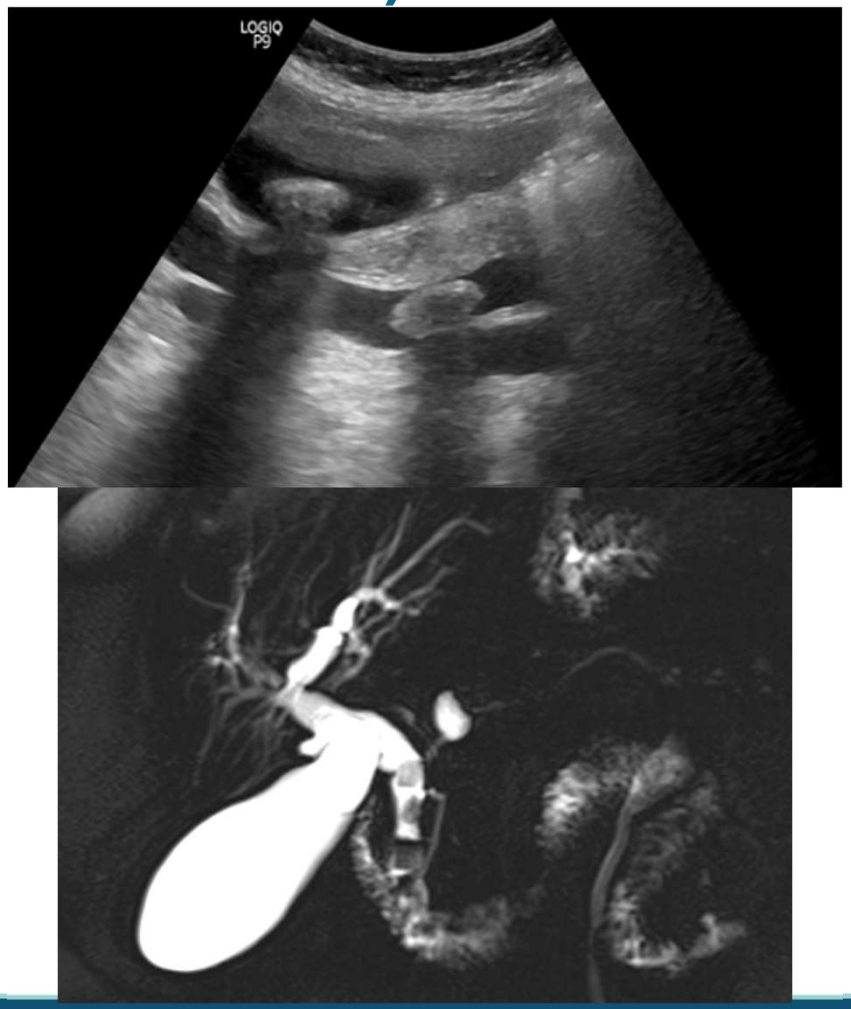
- **Clinical presentations:** acute pancreatitis, biliary colic, ascending cholangitis

What your clinician wants to know

✓ CBD diameter

✓ Presence or absence of gallstones

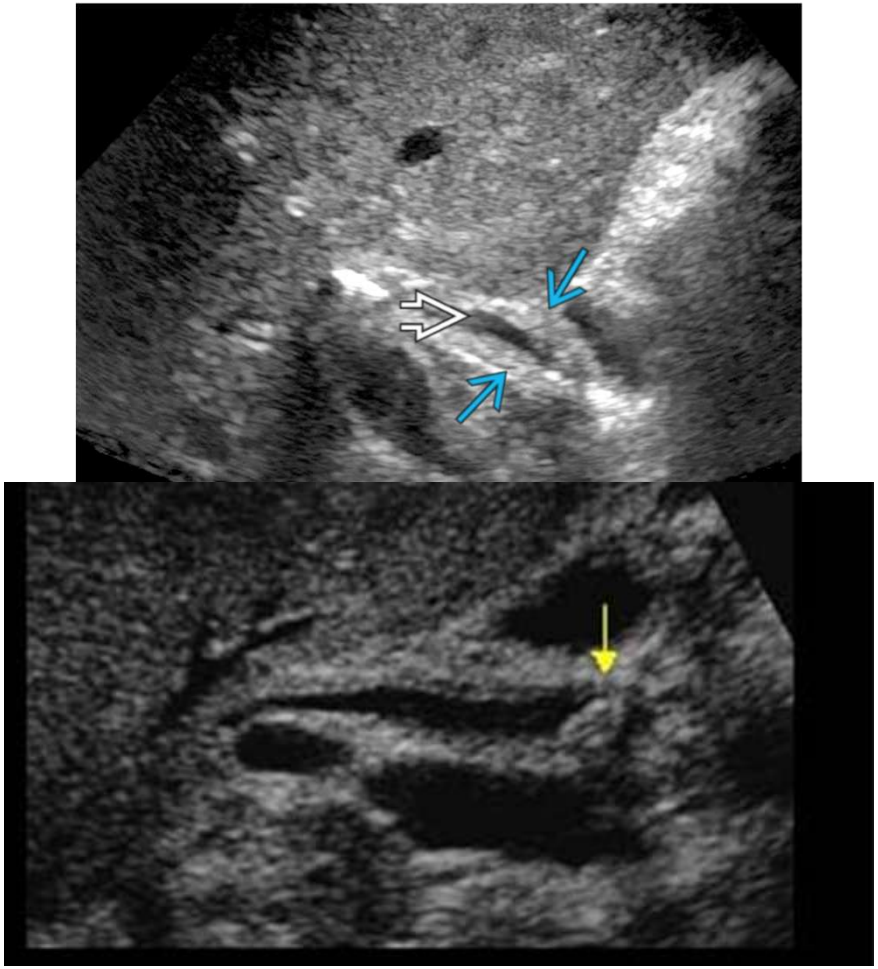
✓ Can you see down to pancreatic head?



Ascending (Acute) Cholangitis

- **Ascending cholangitis** is a bacterial infection of the biliary tree that occurs when **biliary obstruction** and **bacterial colonization** of the bile co-incide.
- Biliary obstruction → forces bacteria and endotoxin into bloodstream → bacteremia and sepsis
- **Surgical/medical emergency.** Needs drainage (PTC, ERCP)
- **Clinical:** Charcot's triad, Reynold's Pentad (40% of patients)

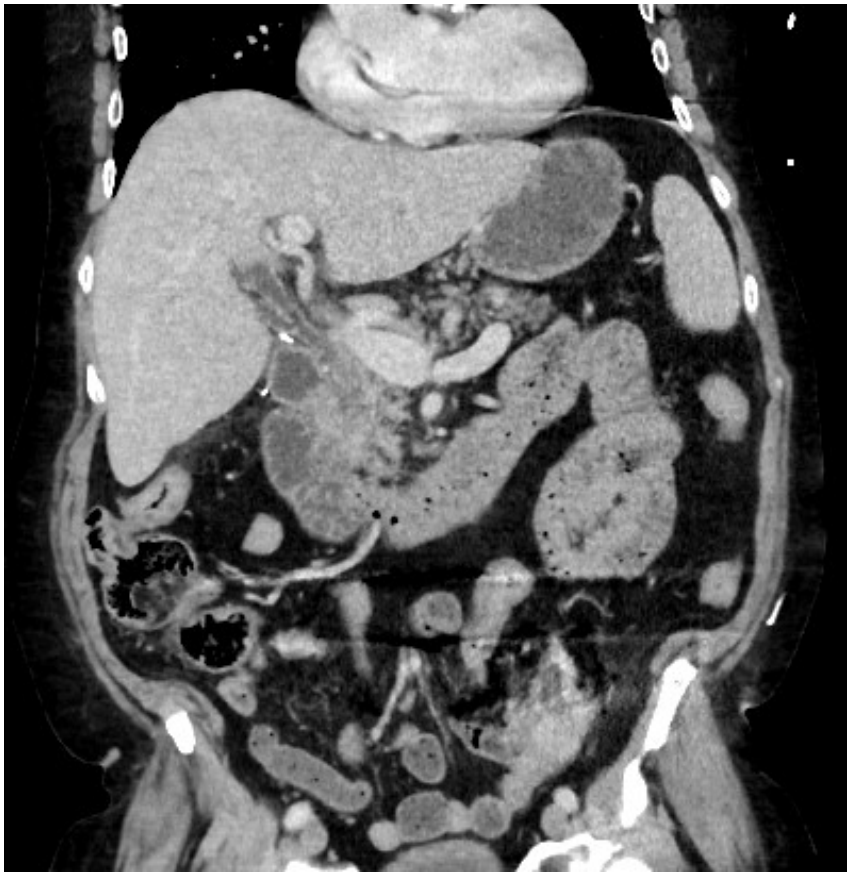
- **On US:** dilated ducts, sometimes with a visible stone, and wall thickening
- **Pneumobilia or debris:** suggests infection or prior intervention
- **Check the parenchyma:** hepatic abscesses can complicate cholangitis
- **Know the limit:** US rules obstruction in but cannot exclude it, so keep a low threshold for MRCP or CT



Ascending (Acute) Cholangitis

What your clinician wants to know:

- ✓ Biliary dilatation – level? Thickening?
- ✓ CBD stones?
- ✓ Cholelithiasis? Cholecystitis?
- ✓ HA/PV patency
- ✓ Complications: hepatic abscess?
- ✓ Pericholecystic fluid, generalized free fluid



Liver Parenchymal abnormalities

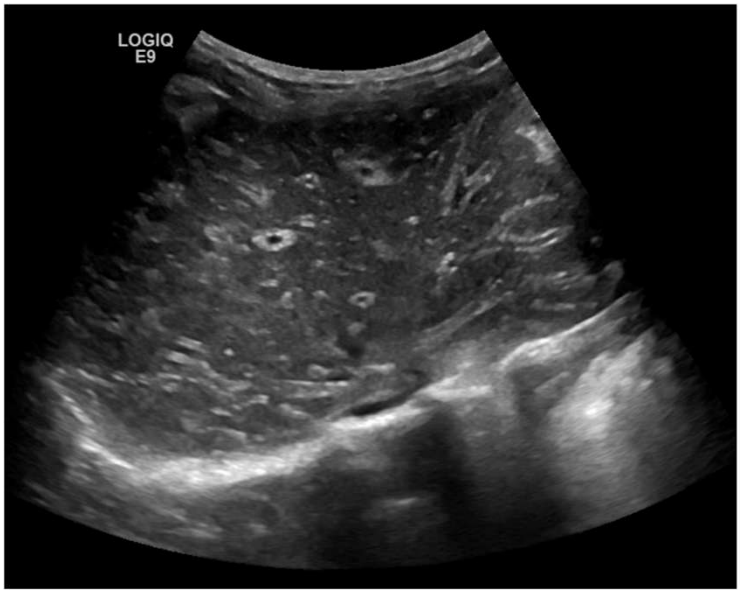
- **Acute hepatitis:** Hepatomegaly, diffuse hypoechogenecity
- **Fatty change:** bright liver, posterior beam attenuation, blurred vessel walls
- **Abscess:** complex hypoechoic collection, sometimes with gas; correlate with sepsis
- **Primary liver lesions:** HCC, intrahepatic cholangiocarcinoma
- **Metastases:** often multiple target lesions distorting the architecture
- **Cirrhosis:** nodular surface, coarse echotexture, signs of portal hypertension
- **Congestion or Budd-Chiari:** distended hepatic veins and IVC, with abnormal Doppler flow

Acute Hepatitis

- Diffuse hepatocyte injury with resulting inflammatory change
- **Ultrasound:** Non-specific features. Hepatomegaly is the most sensitive sign; \
- Starry sky appearances → parenchymal hypoechogenicity because of oedema, resulting in increased prominence of the echogenic portal triads and hepatomegaly

What your clinician wants to know:

- ✓ Hepatomegaly
- ✓ Parenchymal appearances
- ✓ GB wall thickening (more with hepatitis A)
- ✓ Periportal oedema
- ✓ HA/PV patency
- ✓ Other potential causes of liver dysfunction (GB/CBD stones causing cholangitis, liver abscess etc)

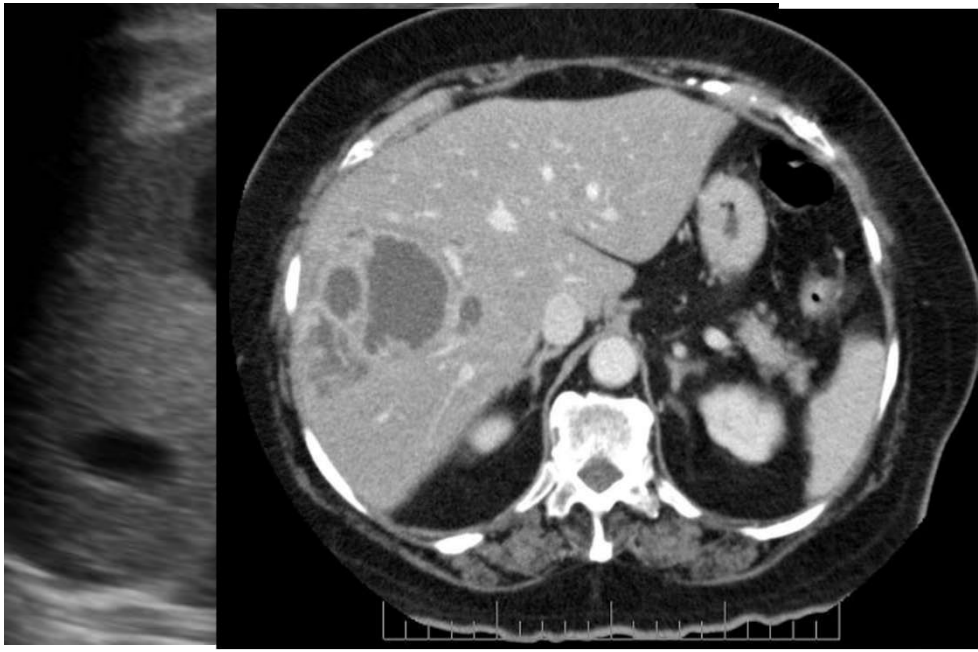


Liver Abscess

1. **Pyogenic:** complex, hypoechoic collection: thick wall, septa, debris +/- gas. Cluster/target sign. Usually biliary or portal source.
2. **Amoebic:** Solitary right lobe, subcapsular, round with low–level echoes. Ask about travel history.
3. **Hydatid:** Well-defined cyst, daughter cysts, detached membrane (water-lily).
4. **Microabscesses:** Multiple tiny lesions; fungal (Candida) if immunocompromised.

What your clinician wants to know:

- ✓ Abscess or tumour? *(Complex cystic vs necrotic solid lesion)*
- ✓ Single or multiple? Size and segment?
- ✓ Is it drainable? *(liquefied and accessible?)*
- ✓ Likely source? *(biliary, portal or hydatid features)*
- ✓ Complications? *(rupture, gas, pleural/subphrenic spread, PV thrombosis)*



Pyogenic liver abscesses

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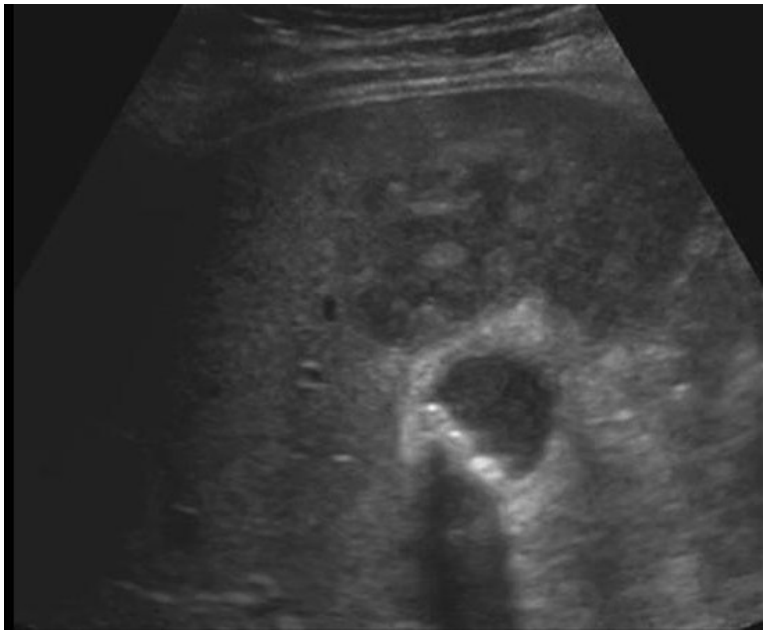

Acute cholecystitis with hepatic abscess

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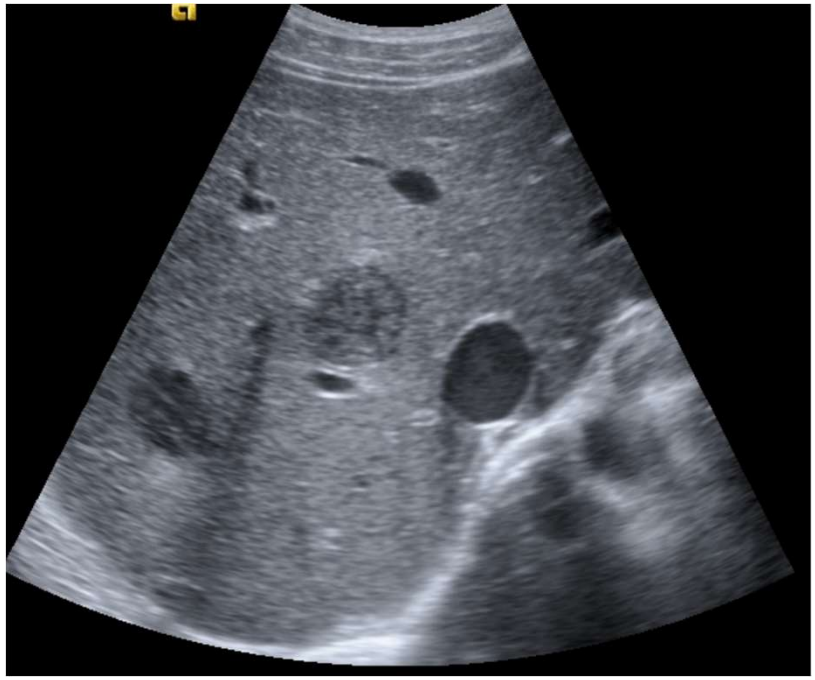

Amoebic abscess

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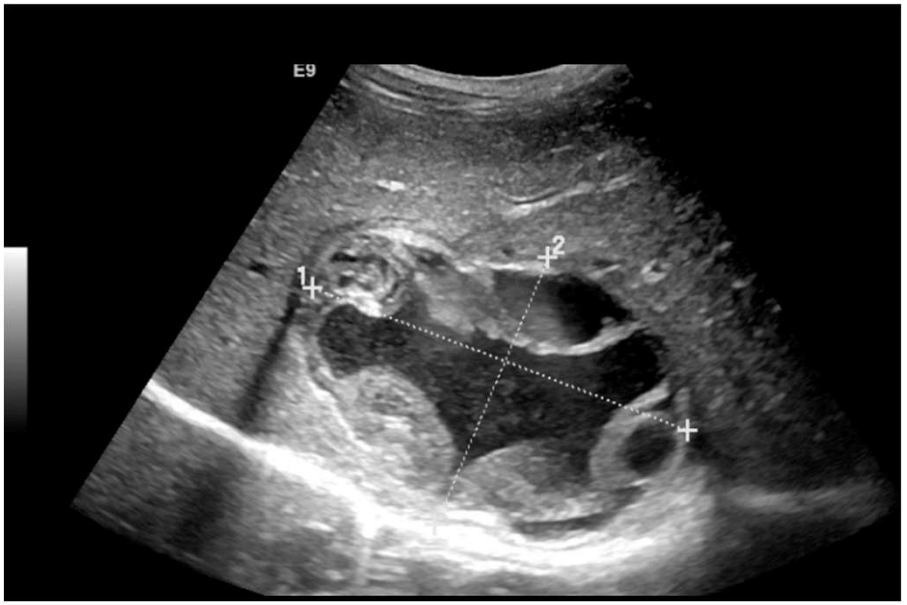

Hydatid Cyst

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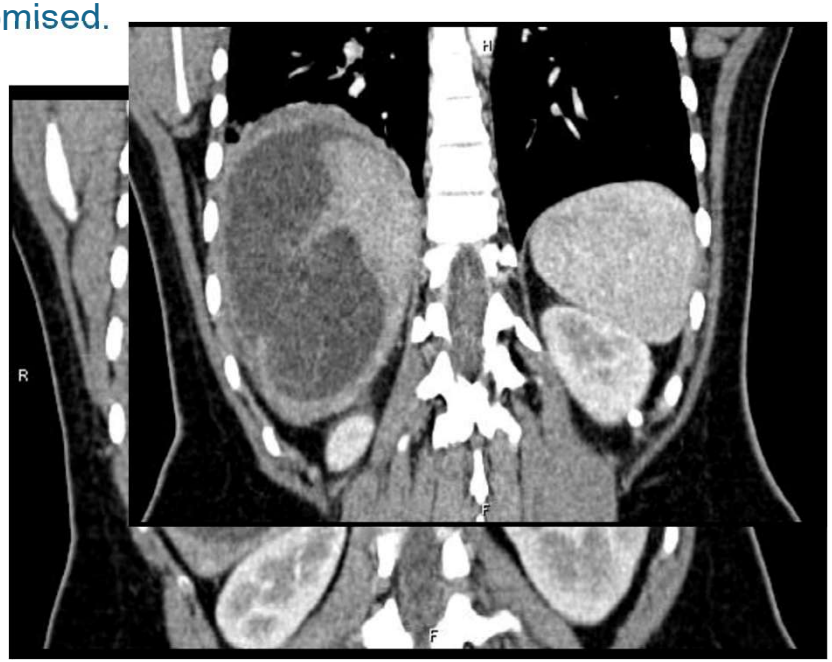


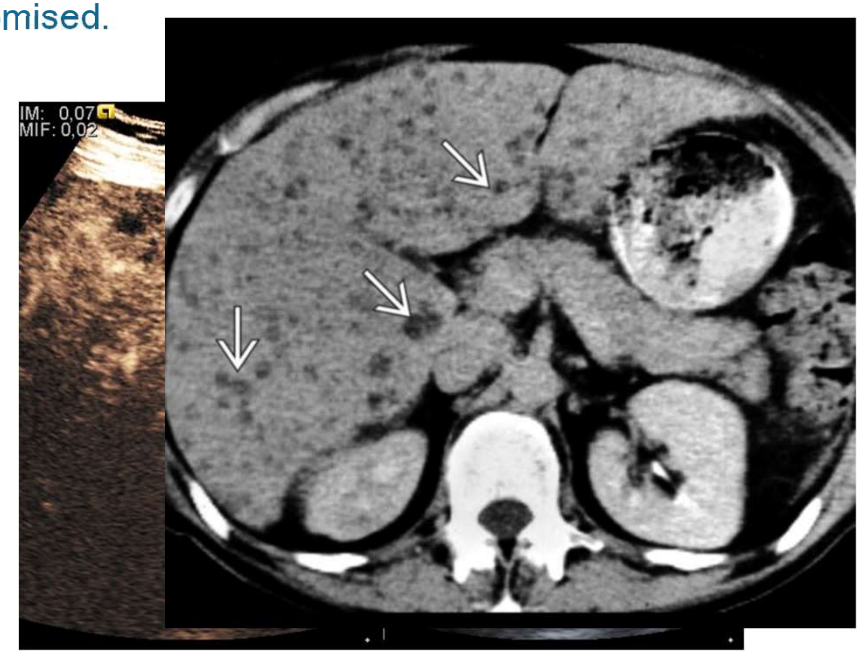
Hydatid Cyst

Liver Abscess

- **Pyogenic:** complex, hypoechoic collection: thick wall, septa, debris +/- gas. Cluster/target sign. Usually biliary or portal source.
- **Amoebic:** Solitary right lobe, subcapsular, round with low–level echoes. Ask about travel history.
- **Hydatid:** Well-defined cyst, daughter cysts, detached membrane (water-lily).
- **Microabscesses:** Multiple tiny lesions; fungal (Candida) if immunocompromised.

What your clinician wants to know:

- ✓ Abscess or tumour? *(Complex cystic vs necrotic solid lesion)*
- ✓ Single or multiple? Size and segment?
- ✓ Is it drainable? *(liquefied and accessible?)*
- ✓ Likely source? *(biliary, portal or hydatid features)*
- ✓ Complications? *(rupture, gas, pleural/subphrenic spread, PV thrombosis)*



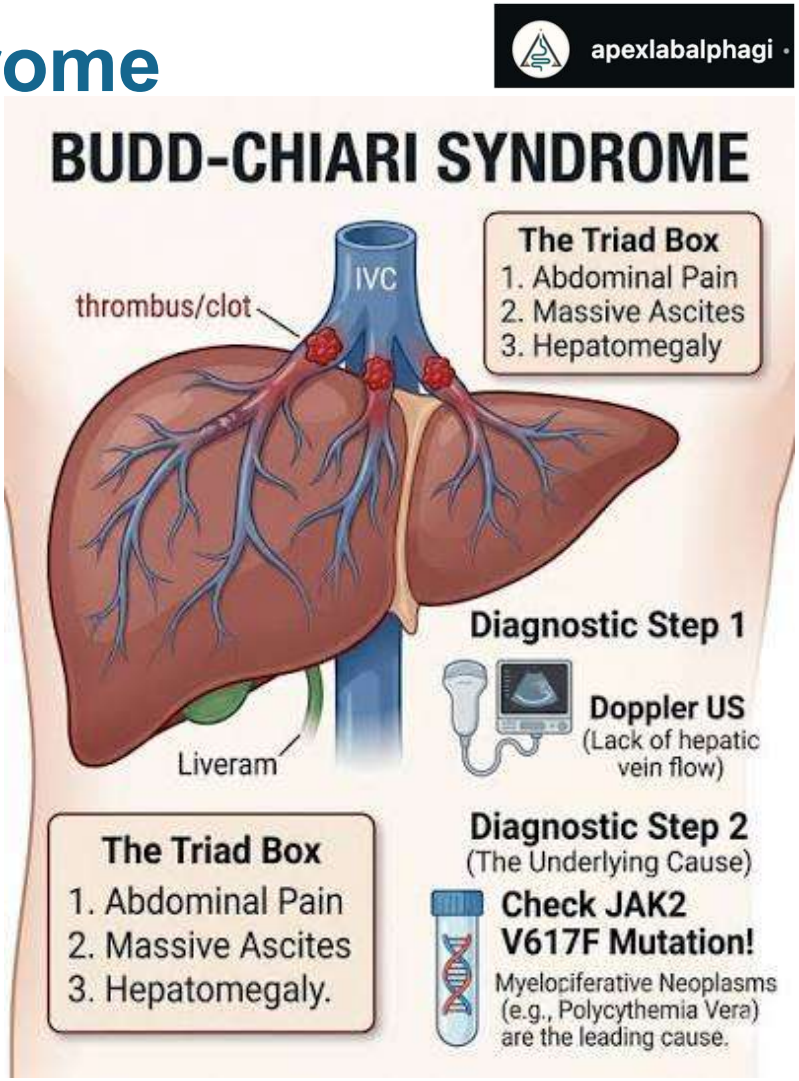
Microabscesses: candidiasis
Ddx: mets, lymphoma, biliary hamartomas, cysts

Budd-Chiari Syndrome

- Partial or complete obstruction of the hepatic veins
- Often an acute on chronic phenomenon
- **Classic clinical presentation:** ascites (often rapid), hepatomegaly, abdominal pain
- **Aetiology:** Idiopathic, IVC/hepatic venous web, pro-thrombotic state

What your clinician wants to know:

- ✓ Patency of each hepatic vein
- ✓ PV patency and direction of flow
- ✓ Hepatic artery RI raised >0;75?
- ✓ Ascites?
- ✓ Hepatomegaly?
- ✓ Liver parenchyma: heterogenous?
- ✓ Caudate hypertrophy?
- ✓ Liver nodules? (FNH-like lesions, regenerative nodules)
- ✓ GB wall thickening?



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https://radiologykey.com/budd-chiari-syndrome/

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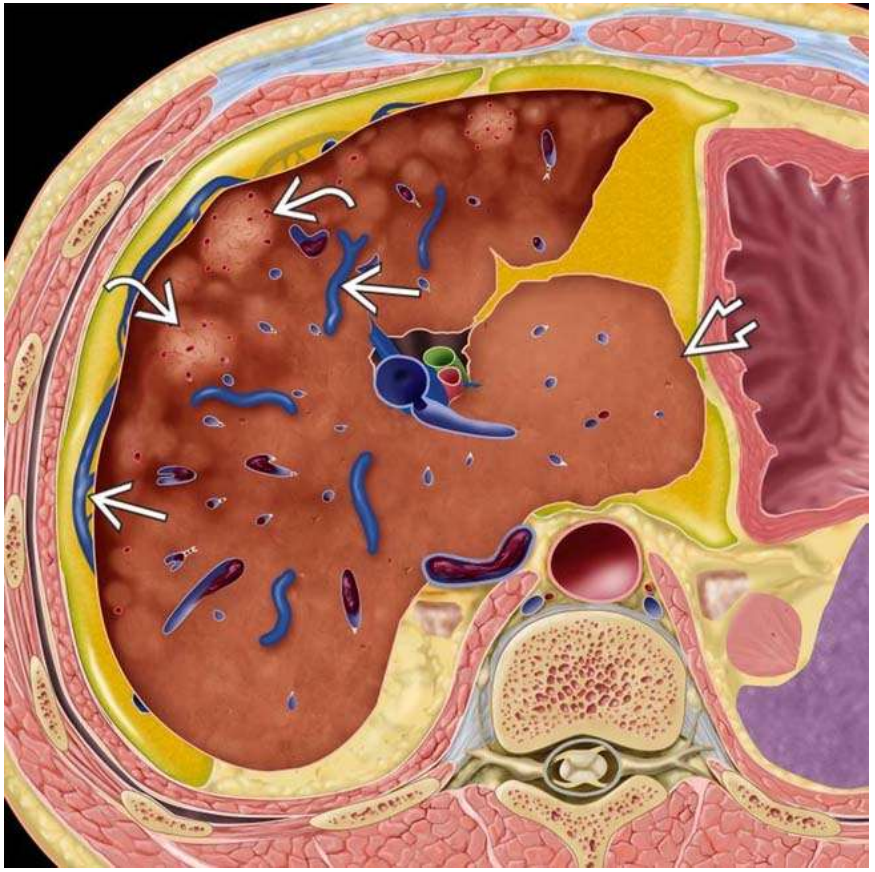

Hepatic venous collaterals, heterogenous parenchyma, caudate hypertrophy

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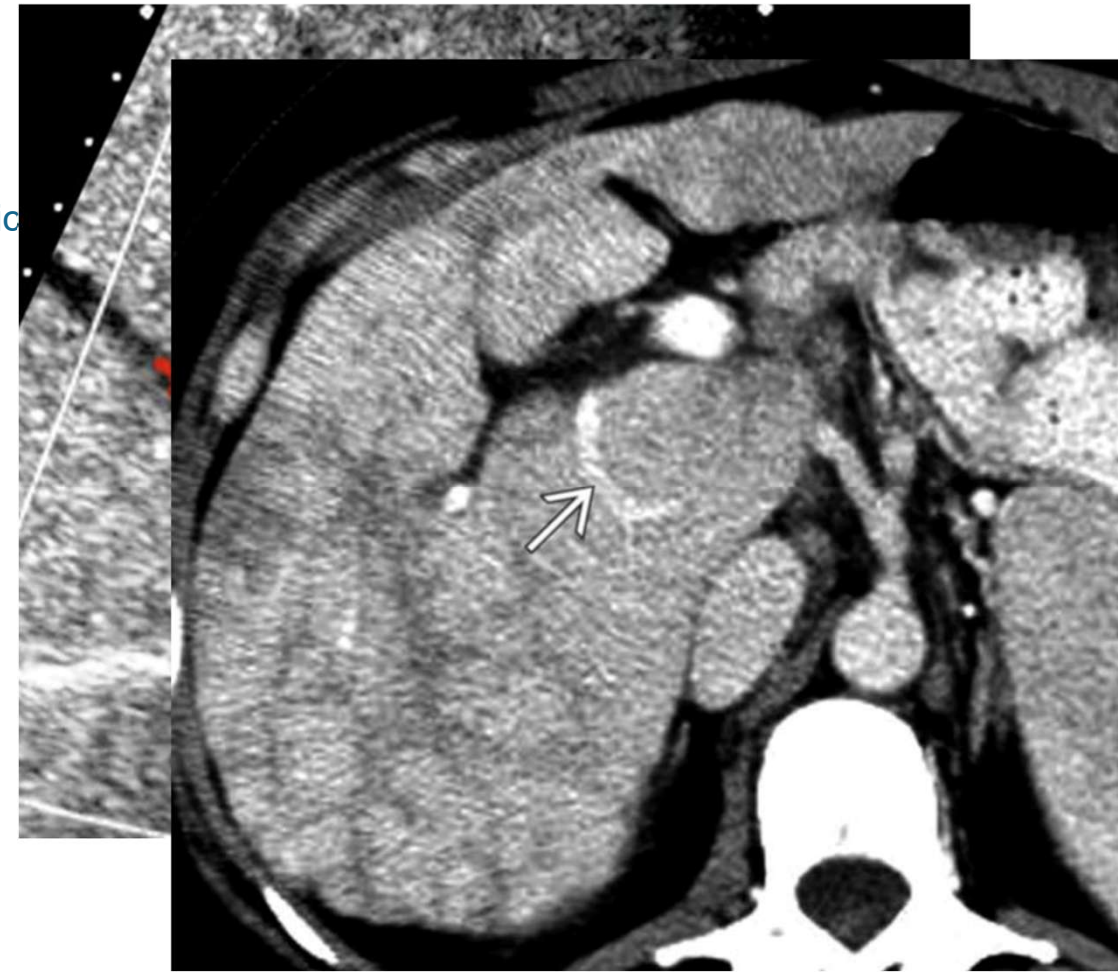

PART 4

Useful vs Useless

Knowing the limits of Ultrasound Imaging in acute HPB imaging

US vs CT vs MRCP

Clinical question	Ultrasound	CT	MRCP
Gallstones / gallbladder	First-line (95%+)	Ok if radio-opaque stones	Good
Acute cholecystitis	First-line	Shows complications	Problem-solving
CBD stones	Misses distal duct	Variable	Gold standard
Duct dilatation & level	Screening tool	Good	Best road-map
Pancreatitis Cause	Gallstone disease	Gold standard for severity + complications	Gallstone disease
Radiation / access	None, bedside	Radiation, fast	None, slower

Ultrasound triages; cross-sectional imaging confirms, stages and maps for intervention.

Role of ultrasound in acute HPB imaging: when is it most clinically appropriate?

WHEN TO PICK ULTRASOUND FIRST No ionising radiation · Bedside & real-time · Detects stones & duct dilatation · Cheap & repeatable

Reach for ultrasound first

- ✓ **RUQ pain — ?acute cholecystitis**
  Stones · wall >3 mm · sonographic Murphy's · pericholecystic fluid
- ✓ **Jaundice or cholestatic LFTs**
  Biliary dilatation? Level of obstruction
- ✓ **Suspected gallstone disease**
  ~95% sensitive for gallbladder stones & sludge
- ✓ **Pregnant, young, or serial scans**
  No ionising radiation; bedside & repeatable

Escalate beyond ultrasound

- ➡ **Distal CBD or stone not seen**
  US sees only ~20–50% of CBD stones — MRCP or EUS
- ➡ **Pancreatitis severity / necrosis**
  Contrast-enhanced CT — US finds the gallstone cause
- ➡ **Staging malignancy**
  CT / MRI
- ➡ **Bowel gas or large body habitus**
  Limits views — cross-sectional imaging

Bottom line — ultrasound is a good first-line test in acute HPB: fast, safe and decisive for gallstones and duct dilatation — with CT, MRCP and EUS reserved for what it cannot resolve.

What Your Clinician Wants to Know

Gallbladder & biliary tree

- **Stones or sludge present?**
  Single, multiple, or impacted
- **Gallbladder acutely inflamed?**
  Wall >3 mm · Murphy's sign · pericholecystic fluid
- **Biliary tree obstructed — at what level?**
  Intrahepatic vs extrahepatic
- **Duct stone, or dilatation only?**
  CBD >6–7 mm — add ~1 mm/decade over 60; wider post-cholecystectomy

Liver

- **Parenchyma normal, fatty, or cirrhotic?**
  Echogenicity · surface nodularity
- **Focal lesion?**
  Mass, cyst, or abscess
- **Size & contour?**
  Hepatomegaly, nodular surface, caudate hypertrophy

Pancreas & other findings

- **Pancreatic head mass?**
  Double-duct sign · distal CBD obstruction
- **Free fluid, ascites, or nodes?**
  Perihepatic / pelvic fluid · porta hepatis lymphadenopathy

Vessels & flow

- **Portal vein patent? Flow direction?**
  Hepatopetal vs reversed; thrombus
- **Hepatic veins & IVC?**
  Patency and waveform
- **Portal hypertension signs?**
  Splenomegaly · ascites · varices · reversed portal flow

Urgent — Biliary sepsis needing decompression? Charcot's / Reynolds' features → escalate for ERCP or PTC (will usually have cross-sectional imaging first).

QUESTIONS & DISCUSSION

Thank You

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