PANCREATITIS AND CHOLECYSTITIS

PANCREATITIS:

- Acute pancreatitis is an acute inflammatory process of the pancreas that involves surrounding tissue and remote organ systems
- The disease ranges from mild inflammation to severe extensive pancreatic necrosis and multi-organ failure with mortality rates of 20-30% or higher
- Common causes listed below → gallstones account for 35-40% of cases and are the most common cause followed by alcohol use (surprisingly, the incidence in alcoholics is low → implicates other factors other than just amount of alcohol consumed)

Table 82-1 Causes of Acute Pancreatitis
Gallstones (including microlithiasis)
Alcohol (acute and chronic alcohol consumption)
Hypertriglyceridemia
Endoscopic retrograde cholangiopancreatography
Drugs
Autoimmune disease (e.g., systemic lupus erythematosus, Sjögren syndrome)
Genetic factors (PRSS1, SPINK1, CFTR)
Abdominal trauma
Postoperative complications (abdominal or cardiac surgery)
Bacterial infections (*Legionella*, *Leptospira*, *Mycoplasma*, *Salmonella*)
Viral infections (mumps virus, coxsackievirus, cytomegalovirus, echovirus, hepatitis B virus)
Parasitic infections (*Ascaris*, *Cryptosporidium*, *Toxoplasma*)
Hypercalcemia
Hyperparathyroidism
Ischemia
Posterior penetrating ulcer
Scorpion venom
Organophosphate insecticide
Pancreatic or ampullary tumor
Pancreas divisum with ductular narrowing on pancreatogram
Oddi sphincter dysfunction
Idiopathic

- About 5% of patients are at risk of develop acute pancreatitis within 30 days of ERCP
- Hypertriglyceridaemia is a rare cause (1-4%) and serum levels of >11 can worsen attacks
- Many drugs are implicated (see below) but they account for only 1.4-2.0% cases

Table 82-2 Drugs Associated with Acute Pancreatitis	
Class I Drugs	**Class II Drugs**
Didanosine	Rifampicin
Asparaginase	Lamivudine
Azathioprine	Octreotide
Valproic acid	Carbamazepine
Pentavalent antimonials	Acetaminophen
Pentamidine	Phenformin
Mercaptopurine	Interferon-α-2b
Mesalamine	Enalapril
Various estrogens	Hydrochlorothiazide
Opiates	Cisplatin
Tetracycline	Erythromycin
Cytarabine	Cyclopenthiazide
Steroids	
Trimethoprim-sulfamethoxazole	
Sulfasalazine	
Furosemide	
Sulindac	

Class I drugs are associated with ≥20 case reports with at least one drug re-exposure, class II 10-20 case reports with/without re-exposure

- Despite advances in diagnostic modalities, about 20% cases remain idiopathic

PATHOPHYSIOLOGY:

- Acute pancreatitis is caused by unregulated activation of trypsin within pancreatic acinar cells → trypsin activates digestive enzymes as well as complement cascade/kinins → autodigestion, pancreatic injury and inflammation
- Local complications include → pseudocyst, acinar cell necrosis and abscess
- The acute inflammatory process itself and the release of mediators from the pancreas and extrapancreatic organs cause REMOTE ORGAN INJURY AND SYSTEMIC INFLAMMATORY RESPONSE SYNDROME, MULTI-ORGAN FAILURE AND DEATH

CLINICAL FEATURES:

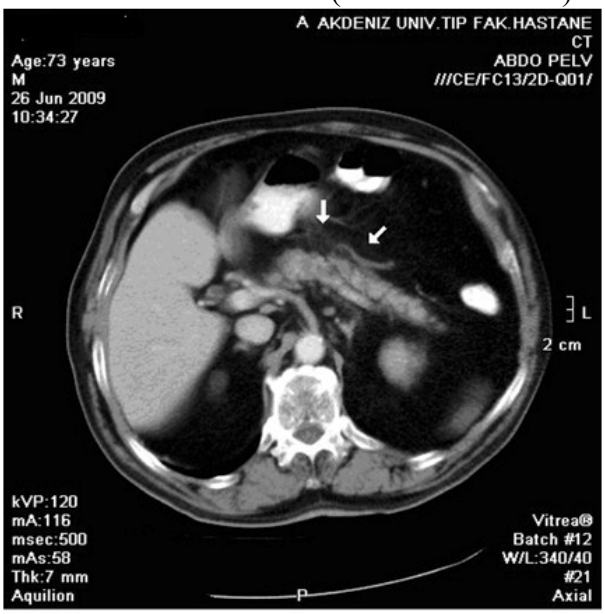
- Typically presents with persistent abdominal pain which often localises to the epigastric area, around the waist, RUQ and occasionally the LUQ
    - Pain may also radiate to the back
    - Can be mild but also causes incapacitating distress
    - Sudden onset abdominal pain reaches maximum intensity in 30 minutes and might last for several days
    - Pain is typically worse in the supine position and can be relieved with sitting up
    - Nausea and vomiting are noted in most
    - Abdominal distension due to GI hypomotility and chemical peritonitis is frequent
- Physical findings depend on the severity of the disease
    - Fever, tachycardia and hypotension are present in severe disease

    - Shock may result form hypovolaemia secondary to:
        - Exudation into the retroperitoneal space
        - Pancreatic haemorrhage
        - Vomiting
        - Increased vasodilation and permeability due to release of kinins
    - Upper abdominal tenderness and guarding is found in variable degreee
    - CULLEN SIGN → echhymoses in periumbilical region
    - TURNER SIGN → echymoses in flanks → Turner and Cullen indicate retroperitoneal and intra-abdominal haemorrhage and severe acute necrotising pancreatitis
    - JAUNDICE → not common and indicates CBD obstruction
- PULMONARY FINDINGS → seen in 10-20% → hypoxia due to ARDS, shallow respiration from diaphragmatic irritation
    - Pleural effusion → small to medium normally. If large, think pancreatico-pleural fistula and can be associated with pseudocyst

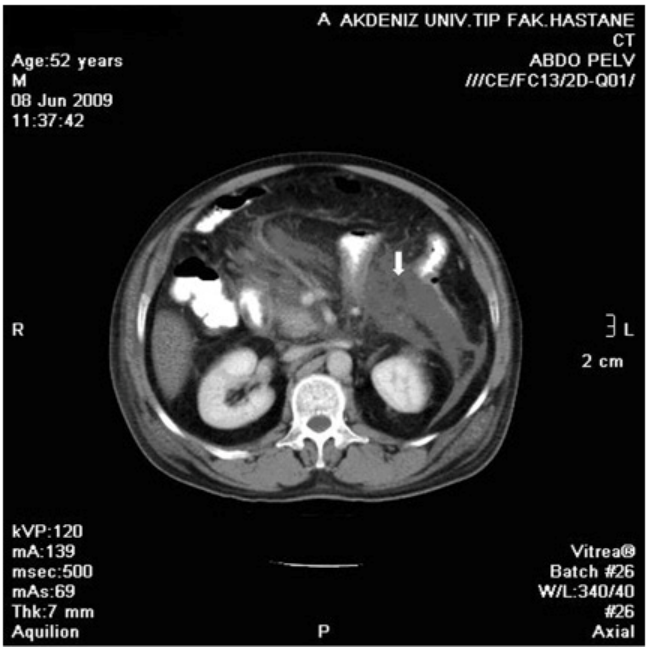
DIAGNOSIS:

- Diagnosis of acute pancreatitis made in the presence of 2 of 3 of the following:
    - Characteristic abdominal pain
    - Serum amylase/lipase levels three times or more the upper limit of normal
    - Characteristic findings on CT or US
- LABORATORY EVALUATION:
    - LIPASE or amylase if lipase not available → need also to take FBC, BSL, LFT, albumin, LDH, EUC, CRP and ABG → used in predicting severity of the disease in prognostic scoring systems, but not in diagnosis
    - A normal amylase is not sufficiently sensitive to exclude acute pancreatitis → but it rises within a few hours after the onset of symptoms and returns to normal within 3-5 days (↑trigs, alcohol reduce sensitivity of amylase)
        - Numerous tissues secrete amylase → can be falsely increased in ↓GFR, diseases of salivary glands, appendicitis, cholecystitis, intestinal obstruction or ischaemia, gynaecologic disease
    - LIPASE → can also be raised in other intra-abdominal pathologies or renal disease but it remains elevated for longer periods and has greater sensitivity
        - A normal lipase does not rule out the diagnosis, especially in recurrent disease
        - IT IS NOT PREDICTIVE OF THE SEVERITY OR OUTCOME OF THE DISEASE
- IMAGING:
    - Plain films not useful, other than for showing ileus or pleural effusions
    - Abdominal US shows an oedematous swollen pancreas → obscuration by gas-filled bowel loops often limit pancreatic imaging by US
        - Can detect underlying structural causes (biliary tree dilation)
    - ABDOMINAL CT is test of choice (especially when history/exam and labs not definitive)

- Excludes alternative diagnoses, determines severity and identify complications
- CT findings range from isolated diffuse or focal enlargement of the gland to peripancreatic stranding and peripancreatic fluid collections and (at its most severe) → pancreatic gland necrosis



Mild pancreatitis with borders of gland becoming indistinct, hazy soft tissue stranding surrounding the pancreas



Acute pancreatic necrosis with small zone of low attenuation representing nonenhancing parenchyma in the pancreatic tail

- Pancreatic necrosis may not be fully apparent on CT for up to 3 days after the disease onset
- ERCP is a diagnostic and therapeutic option for obstructing bile duct and pancreatic duct lesions
    - ERCP has several DISADVANTAGES → bleeding post-sphincterotomy, as well as itself can cause acute pancreatitis

- Urgent ERCP and biliary sphincterotomy are indicated for patients with severe biliary pancreatitis with retained CBD stone and for those with cholangitis

COMPLICATIONS OF PANCREATITIS:

Table 82-3 Complications of Acute Pancreatitis

Local	Systemic
Pancreatic necrosis	Cardiovascular
Sterile	Hypotension
Infected	Hypovolemia
Pancreatic fluid collection	Myocardial depression
Pancreatic abscess	Pericardial effusion
Pancreatic pseudocyst	Pulmonary
Pleural effusion and fistula	Hypoxemia
Pancreatic ascites	Atelectasis
Involvement of peripancreatic tissues with necrosis	Pleural effusion
	Pulmonary infiltrates
Hemorrhage	Acute respiratory distress syndrome
Pancreatic pseudocyst	Respiratory failure
Pseudoaneurysm	Hematologic
Thrombosis—splenic vein, portal vein	Disseminated intravascular coagulation
Bowel infarction	GI
Biliary obstruction with jaundice	Peptic ulcer disease/erosive gastritis
	GI perforation
	GI bleeding
	Obstruction of duodenum or stomach
	Inflammation of the transverse colon
	Splenic infarct
	Renal
	Oliguria
	Azotemia
	Acute renal failure
	Renal artery and/or vein thrombosis
	Metabolic
	Hyperglycemia
	Hypocalcemia
	Hypertriglyceridemia

ARDS, renal failure, shock, encephalopathy and intraabdominal haemorrhage are seen early (first few days). Infectious complications are seen 5-15 days, abscess >15 days post onset

PREDICTION OF SEVERE ACUTE PANCREATITIS:

- Severe acute pancreatitis is defined as the presence of organ failure or local complications such as necrosis, abscess or pseudocyst
  - Assessment of severity is crucial for disposition, because severe cases are associated with higher mortality and morbidity
- CLINICAL AND LAB SCORING SYSTEMS:
  - Glasgow score

Assessing the severity of acute pancreatitis

Glasgow prognostic score: (NOTE PANCREAS ACRONYM)

- **P**aO2 < 8kPa (60mmhg)
- **A**ge > 55 years
- **N**eutrophils: (WBC >15 x109/l
- **C**alcium < 2mmol/l
- **R**enal function: (Urea > 16mmol/l)
- **E**nzymes: (AST/ALT > 200 iu/L or LDH > 600 iu/L)
- **A**lbumin < 32g/l
- **S**ugar: (Glucose >10mmol/L)

Any 3 factors means acute severe pancreatitis

- **RANSON CRITERIA:**
  - 11 point scoring system is applied in two stages
    - 5 initial data points on admission
    - 6 further data points obtained within subsequent 48 hours
    - Less useful in ED setting due to subsequent measures involved in scoring

Table 82-4 Ranson Criteria*

At Admission	Within Next 48 h
Age >55 y (>70 y)	Decrease in hematocrit by >10% (same)
White blood cell count >16,000/mm^3 (>18,000/mm^3)	Estimated fluid sequestration >6 L (>4 L)
Blood glucose level >200 milligrams/dL (>220 milligrams/dL)	Serum calcium level <8.0 milligrams/dL (same)
Serum lactate dehydrogenase level >350 IU/L (>400 IU/L)	Partial pressure of arterial oxygen <60 mm Hg (omitted)
Serum aspartate aminotransferase level >250 IU/L (same)	Increase in blood urea nitrogen level >5 milligrams/dL after IV fluid hydration (>2 milligrams/dL)
	Base deficit of >4 mmol/L (>6 mmol)

- **CT SCORING SYSTEMS OF SEVERITY:**
  - Sums amount of necrosis and severity of pancreatitis

Table 82-5 CT Severity Index for Acute Pancreatitis		
Grade of Acute Pancreatitis		**Score**
Normal pancreas		0
Pancreatic enlargement		1
Inflammation involving pancreas and peripancreatic fat		2
Single fluid collection or phlegmon		3
Two or more fluid collections or phlegmons		4
Degree of Pancreatic Necrosis		**Score**
No necrosis		0
Necrosis of one third of pancreas		2
Necrosis of one half of pancreas		4
Necrosis of more than one half of pancreas		6
Interpretation (minimum score = 0 and maximum score = 10)		
Severity Index	Mortality (%)	Complications (%)
0–1	0	0
2–3	3	8
4–6	6	35
7–10	17	92

- Risk factors predicting the severity of acute pancreatitis at admission include some clinical predictors and laboratory measures representing organ failure:
    - Older age → >55
    - Obesity → BMI > 30
    - Haematocrit ≥44%
    - Presence of shock (SBP <90)
    - Presence of pulmonary insufficiency (PAO2 ≤60mmHg)
    - Presence of renal failure (creatinine ≥200 after hydration)
    - GI bleeding
    - DIC (platelets <100, fibrinogen <1)
    - Severe metabolic disturbances (hyperglycaemia, hypocalcaemia)

TREATMENT OF ACUTE PANCREATITIS:

- If mild → supportive care with close monitoring of haemodynamic and volume status, adequate fluid resuscitation, correction of electgrolyte and metabolic imbalances, effective pain control, bowel rest
    - Ensure early diagnosis of severe cases
- Hypocalcaemia is common and is most often related to low albumin → unless IONISED CALCIUM IS LOW OR PATIENT IS SYMPTOMATIC, DO NOT REPLACE CALCIUM
- In general, routine use of antibiotics or antifungals is NOT supported by the evidence → should be given for infected pancreatic necrosis, pancreatic abscess or infected peripancreatic fluid collection or infected pancreatic pseudocyst
    - Antibiotic chosen should be one with adequate penetration into necrotic material → IMIPENEM, MEROPENEM OR COMBINATIN OF FLUOROQUINOLONE AND METRONIDAZOLE

- ERCP → for those with severe acute biliary pancreatitis with retained CBD stone and those with cholangitis → reduced complications BUT DOES NOT IMPROVE MORTALITY
    - In those with biliary pancreatitis, cholecystectomy indicated during the index visit or shortly thereafter to prevent recurrent episodes
    - Early surgery (<14 days) should be avoided unless there are specific indications, as early surgery is associated with increased mortality
        - Indicated in those who have a catastrophic complication of pancreatitis → haemorrhage, bowel infarction, abdominal comprtment syndrome or perforation
- Large, symptomatic or complicated pseudocysts can be managed with surgical, radiologic and endoscopic options → endoscopic ultrasound is becoming more prevalent
- General measures outlined below:

Table 82-6 Treatment of Acute Pancreatitis

Treatment	Comments
Vital sign measurement and pulse oximetry	Take readings every 2–4 h for first 24 h.
Aggressive crystalloid therapy	Maintain urine at 0.5 mL/kg as long as no renal failure; avoid hypovolemia, which may further diminish microvascular blood flow to pancreas.[1,132–134]
Monitoring of hematocrit, glucose level, calcium level, albumin level	Measure ionized calcium and treat if symptomatic hypocalcemia[56]; control hyperglycemia; administer packed red blood cells if needed to maintain hematocrit; consider administration of albumin if level <2 grams/dL.
Parenteral narcotics	Control pain.
Antiemetics	Control nausea and vomiting; place on NPO status; nasogastric suction typically not indicated.[56,135]
Antibiotics	For abscess, infected pseudocyst, infected peripancreatic fluid, give imipenem-cilastatin, meropenem, or a combination of a fluoroquinolone and metronidazole. Prophylactic antibiotics and antibiotics for mild pancreatitis not indicated.
Consultation for endoscopic retrograde cholangiopancreatography with biliary sphincterotomy	In first 24 h for those with retained common duct stones or cholangitis.

DISPOSITION:

- Patients with mild pancreatitis, no biliary tract disease and no evidence of systemic complications can be discharged home if they can tolerate oral liquids and if pain is adequately controlled
- All other patients must be admitted
- Patients with SAP with SIRS, sepsis, severe comorbid conditions, hypoxia or renal insufficiency as well as those requiring aggressive fluid resuscitation need ICU input

CHOLECYSTITIS:

- A class of related disease states with different causes
- CHOLECYSTITIS:
  - Inflammation of the gallbladder, most often due to gallstones
  - In acute cholecystitis, if bacterial invasion occurs after inflammation → COMPLICATIONS → ascending cholangitis, empyema, emphysematous cholecystitis can result
  - BILIARY COLIC → syndrome of constant RUQ pain lasting 2-6 hours, remits spontaneously
- ACUTE ACALCULOUS CHOLECYSTITIS:
  - Develops in the absence of gallstones
  - Major RF include → burns, old age, critical illness, trauma, major surgery, long term TPN, DM, immunosuppression and child birth
- GALLBLADDER PERFORATION:
  - Seen in 10% patients with acute cholecystitis
  - Perforation into a hollow viscus can lead to GALL-STONE ILEUSas a result of CHOLECYSTIC-ENTERIC FISTULA
  - If contents spill into peritoneum → peritonitis
- CHRONIC CHOLECYSTITIS:
  - Protracted GB inflammation with progressive fibrotic thickening of the gallbladder wall from repeated injury, almost always from gallstones
- EMPHYSEMATOUS CHOLECYSTITIS:
  - Infection of the GB wall caused by gas-forming organisms
  - Can lead to rapid clinical deterioration with mortality as high as 15% because of gangrene or perforation
- CLINICALLY SILENT GALLSTONES:
  - I.E. ASYMPTOMATIC → once diagnosed, the risk of developing pain or complications is low → 1-4% per year with only 10-20% developing symptoms with 5 and 20 years respectively
- CHOLANGITIS:
  - Ascending infection due to partial or complete bile duct obstruction
- CHOLEDOCHOLITHIASIS:
  - Condition of stones in the CBD

PATHOPHYSIOLOGY:

- Bile is composed mostly of water (80%) as well as bile acids (10% and other phospholipids (4-5%) and cholesterol (15)
- Main function of the GB is to concentrate bile by absorption of water and sodium and empty into the duodenum in response to a fatty meal
- Subjects with large fasting/residual GB volumes may be at increased risk for gallstone disease
- GALLSTONE COMPOSITION:

Table 82-7 Gallstone Composition

Composition	Subcategory	Comment
Cholesterol stones (radiolucent)		
Cholesterol monohydrate crystals (70%)	—	Bile supersaturation with hepatically generated cholesterol and biliary stasis leading to formation of biliary sludge
Pigmented stones (radiopaque)		
Pure pigment stones (20%); mixed stones (10%)	Black stones	Bilirubin polymers—hemolysis, advanced age, alcoholism, pancreatitis, cirrhosis, total parenteral nutrition
	Brown stones	Calcium bilirubinate within gallbladder and intrahepatic and extrahepatic ducts—enteric infection and parasites, associated with cholangitis and biliary sludge

- When gallstones pass through the biliary tract, pain, N+V occur due to increased intraluminal pressure after stone migration into either the cystic or CBD
- If initial obstruction is not relieved by persisten contractions of the biliary system, the inflammatory response occurs by MECHANICAL, CHEMICAL AND INFECTIOUS MEANS:
    - MECHANICAL:
        - Increased intraluminal pressure and hollow viscus distension → VISCERAL ISCHAEMIA
    - CHEMICAL:
        - Causes direct mucosal injury and resultant inflammation
    - INFECTIOUS:
        - Infectious agents identified include gram-negative bacteria, which are present in 74% cases
        - Polymicrobial infection is common (E coli, Kelbsiella, plus gram positives enterococcus, staph and strep, clostridium in some).

CLINICAL FEATURES:

- Acute GB disease should be considered in anyone with upper abdominal pain who still has their GB! → especially the elderly
    - Stones can cause symptoms months to years after cholecystectomy, although the incidence is low

- Classic RUQ pain results from contact of distended GB with peritoneum and pain may radiate to other areas of the abdomen/back/right shoulder
- Pain is not necessarily related to meals or fatty food
- CIRCADIAN RHYTHM with peak of symptoms at midnight to 1am and majority occurring from 9pm to 4am.
- Associated symptoms:
    - NAUSEA, VOMITING, DIAPHORESIS, FEVER
- Biliary colic generally arises when an obstructing stone causes sudden distention of the gallbladder → defined as pain lasting 1-5 hours then remitting
    - Pain resolves if stone spontaneously returns into the gallbladder lumen or passes through the ampulla into the duodenum and if infection has not occurred
    - If pain lasts >5 hours, suspect complications such as acute cholecystitis, ascending cholangitis or pancreatitis

DIAGNOSIS OF CHOLECYSTITIS:

- Depends on combination of findings from history, physical exam and lab/imaging findings
- No single clinical or laboratory finding has sufficiently high LR+ or low LR- to rule in or rule out the diagnosis of acute cholecystitis (see below)

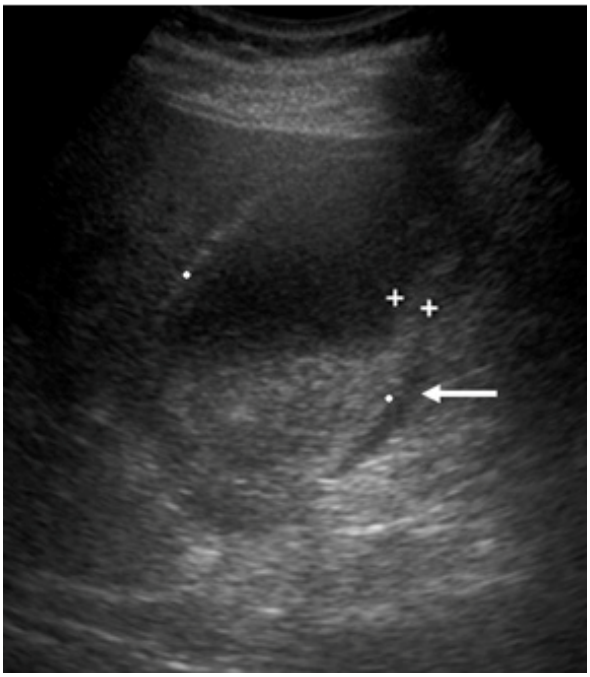
Table 82-8 Summary of Test Characteristics for Selected Clinical and Laboratory Findings in Acute Cholecystitis

Findings	Sensitivity (%)	Specificity (%)	LR+	LR-
Clinical				
Fever	35	80	1.5	0.9
Nausea	77	36	1.5	0.9
Emesis	71	53	1.5	0.6
RUQ pain	81	67	1.5	0.7
RUQ tenderness	77	54	1.6	0.4
Murphy sign	65	87	2.8	0.5
Laboratory				
Leukocyte count >10,000/mL	63	57	1.5	0.6
AP level >120 units/L	45	52	0.8	1.1
Elevated AST or alanine aminotransferase level	38	62	1.0	1.0
TB level >2 milligrams/dL	45	63	1.3	0.9
Elevated level of AP, AST, or TB	70	42	1.2	0.7

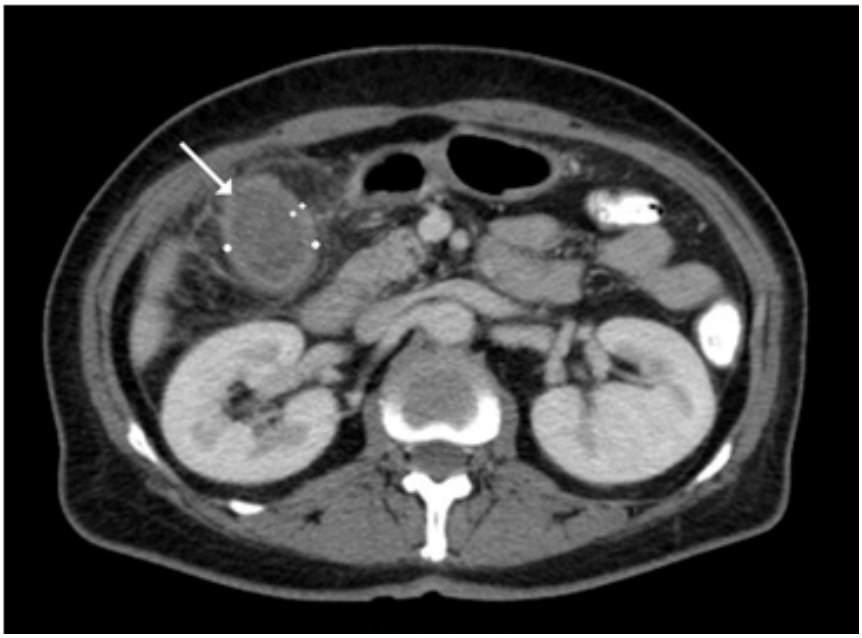
- MURPHY'S SIGN → sudden cessation of deep inspiration due to pain when the examiners hand/finger contacts the inflamed GB during palpation has the highest sensitivity for cholecystitis
- An elevated AST, ALT, ALP should raise the possibility of CBD stone, ascending cholangitis or MIRIZZI SYNDROME (extrinsic compression of the common hepatic duct from an impacted stone in the cystic duct)
- One set of consensus criteria for diagnosis are shown below:

Table 82-9 Diagnostic Criteria for Acute Cholecystitis*	
Local signs	Murphy sign
	Right upper quadrant mass, pain, tenderness
Systemic signs of inflammation, etc.	Fever
	Elevated C-reactive protein level
	Elevated white blood cell count
Imaging	Gallbladder wall >3 mm thick†
	Pericholecystic fluid
	Biliary duct diameter >7 mm†

- **IMAGING:**
  - Plain radiography is poor (only 20% of stones are radioopaque)
  - RUQ SONOGRAPHY:
    - Imaging modality of choice with sensitivity and specificity of 94% and 78% respectively for detecting acute cholecystitis
    - Not as good at detecting stones ≤1mm due to poor echogenicity and stones impacted at the GB neck or cystic duct can be difficult to detect on US
    - SONOGRAPHIC MURPHY'S SIGN → presence of maximal tenderness over the sonograhically identified gallbladder → has PPV of 92% in conjunction with the presence of gallstones and PPV of 87% in ocnjunction with other clinical findings
      - May not be present in diabetics or those with gangrenous cholecystitis
    - Cholecystitis should be considered in those with wall thickness ≥3mm, but this can also occur with the following:
      - Pancreatitis
      - Ascites
      - Right heart failure
      - Alcoholic hepatitis
        - Most patients with cholecystitis have values ≥5mm
    - Pericholecystic fluid quite specific for cholecystitis
    - CBD diameter (upper limit normal is 7mm)
    - See image below
  - CT → USEFUL WHEN US EQUIVOCAL
    - May have sensitivity, specificity up to 95% in presence of four criteria
      - Wall thickness >3mm
      - Short axis dimension of >3.5cm
      - Presence of stones
      - Presence of pericholecystic fluid
    - See image below



Pericholecystic fluid in acute cholecystitis



Enlarged GB with fluid, wall thickening and enlarged short-axis dimension

- TECHNETIUM-99 HEPATOBILIARY IMINODIACETIC ACID CHOESCINTIGRAPHY (HIDA SCAN) evaluates GB function → can identify biliary dyskinesia in setting of other imaging being normal
    - Generally not practical in the ED setting
    - Morphine may interfere with the scan
- MRCP → highest sensitivity for wall thickness and presence of pericholecystic fluid
- Findings of various forms of imaging summarised below:

Table 82-10 Imaging Findings of Acute Cholecystitis
US Findings
Primary
Sonographic Murphy sign[120]
(PPV 92% with the presence of gallstones)[136]
(PPV 87% with right upper quadrant pain, fever, and elevated white blood cell count)[136]
Secondary[122,136]
Gallbladder wall thickening (>3 mm), striations, layering
(With acute cholecystitis: present in acute gangrenous cholecystitis)[137]
(Without acute cholecystitis: may be present in other clinical diseases)
Pericholecystic fluid[137]
Type I (nonspecific, associated with ascites or other localized inflammatory conditions)
Type II (associated with gallbladder perforation or abscess formation)
Enlarged gallbladder[123]
(Short-axis dimension >4 cm, long-axis dimension >8 cm)
(Presence of gallstones, presence of fluid, debris echo)
CT Findings[123]
Increased gallbladder wall thickness (>3 mm)
Pericholecystic fluid collection
Enlarged gallbladder
(Short-axis dimension >3.5 cm)
(Presence of gallstones, presence of fluid)
Technetium 99m Hepatobiliary Iminodiacetic Acid Cholescintigraphy Findings[138]
Nonvisualized gallbladder with normal uptake and excretion of radioactive tracer, ejection fraction <35%
Rim sign (augmentation of radioactivity around gallbladder fossa)
MRI Findings[124]
Increased gallbladder wall thickness
Pericholecystic fluid collection
Adjacent fat signal

TREATMENT:

- No treatment is required for asymptomatic gallstones unless there are RF for complications (cirrhosis, portal HT, sickle cell disease, transplant candidate)
- Biliary colic → pain is treated with opiates, NSAIDS and OP referral to surgeons for definitive management (i.e. laparoscopic cholecystectomy)
- Acute cholecystitis → hospital admission under surgery for cholecystectomy
    - ERCP undertaken with CBD stones or dilated bile ducts
    - Untreated acute cholecystitis may lead to severe complications → ascending cholangitis, emphysematous cholecystitis, gangrenous cholecystitis, pancreatitis
    - ED treatment → effective analgesia, antiemetics, NBM, volume/electrolyte replacement, IV antibiotics and surgical consult
        - All narcotics increase biliary pressure and induce spasm in the Sphincter of Oddi
        - IV antibiotics → third generation cephalosporin and metronidazole or fluoroquinolone and metronidazole
        - Broader coverage with meropenem or tazocin for those with severe toxicity, comorbidities (immunosuppression or diabetes, advanced age)

SPECIAL POPULATIONS:

- Gallstones and biliary colic in children are uncommon but can occur with congenital anomalies, biliary anomalies, haemolytic states, sickle cell anaemia
- Diabetics and elderly patients have higher morbidity and mortality from cholecystitis or its complications

SPECIAL CONSIDERATIONS:

- ACUTE CHOLANGITIS:
    - Requires presence of biliary obstruction and infected biliary tract
    - Causes of biliary obstruction include:
        - Choledocholithiasis
        - Billiary tract strictures
        - Strciture of a biliary anastomosis
        - Compression caused by malignant disease
    - Classic description is CHARCOT TRIAD:
        - Fever
        - Jaundice
        - RUQ pain
            - Seen in fewer than half patients with cholangitis
    - ED treatment is aggressive fluid resuscitation, IV antibiotics and emergent decompression of biliary tract (ERCP)
- CHRONIC CHOLECYSTITIS:
    - Symptoms and exam ares similar to those of biliary colic and acute cholecystitis but with lower intensity but longer duration of symptoms
- EMPHYSEMATOUS CHOLECYSTITIS:

    - Characterised by air in the GB wall due to infection with gas-forming anaerobes, including Clostridium perfringens
    - This form is likely to progress to sepsis an dgangrenous cholecystitis and is often seen in DIABETICS
- GALLSTONE ILEUS:
    - Bowel obstruction due to impaction of a gallstone at the terminal ileum
    - Gallstone enters the small bowel through a biliary-duodenal fistula
    - Diagnosis is suggested by presence of pneumobilia, bowel obstruction and ectopic gallstone
    - Morbidity and mortality are high