Acute Pancreatitis

Dr. Sachin Naik

ROADMAP

Learning Objectives

Define & classify

Define acute pancreatitis and apply the revised Atlanta classification.

Surgical anatomy

Correlate pancreatic anatomy and relations with disease behaviour.

Etiology & pathogenesis

List causes and explain the mechanism of acinar cell injury.

Clinical assessment

Recognise clinical features and assess severity using scoring systems.

Management

Outline medical management and indications/options for surgery.

Prognosis

Discuss complications, prognosis, and prevention of recurrence.

OVERVIEW

Definition & Epidemiology

Definition

Acute pancreatitis is an acute inflammatory process of the pancreas, arising from premature activation of digestive enzymes within acinar cells, with variable involvement of peripancreatic tissue and remote organs.

Diagnosis — 2 of 3 required

- ✦ Characteristic upper abdominal pain
- ✦ Serum amylase/lipase > 3x upper limit of normal
- ✦ Characteristic findings on CT / MRI / USG

13–45 per 100,000 annual incidence

~80% mild, self-limiting disease

~20% severe disease with organ failure

1st / 2nd Gallstones & alcohol — leading causes

Location & Parts of the Pancreas

Retroperitoneal organ

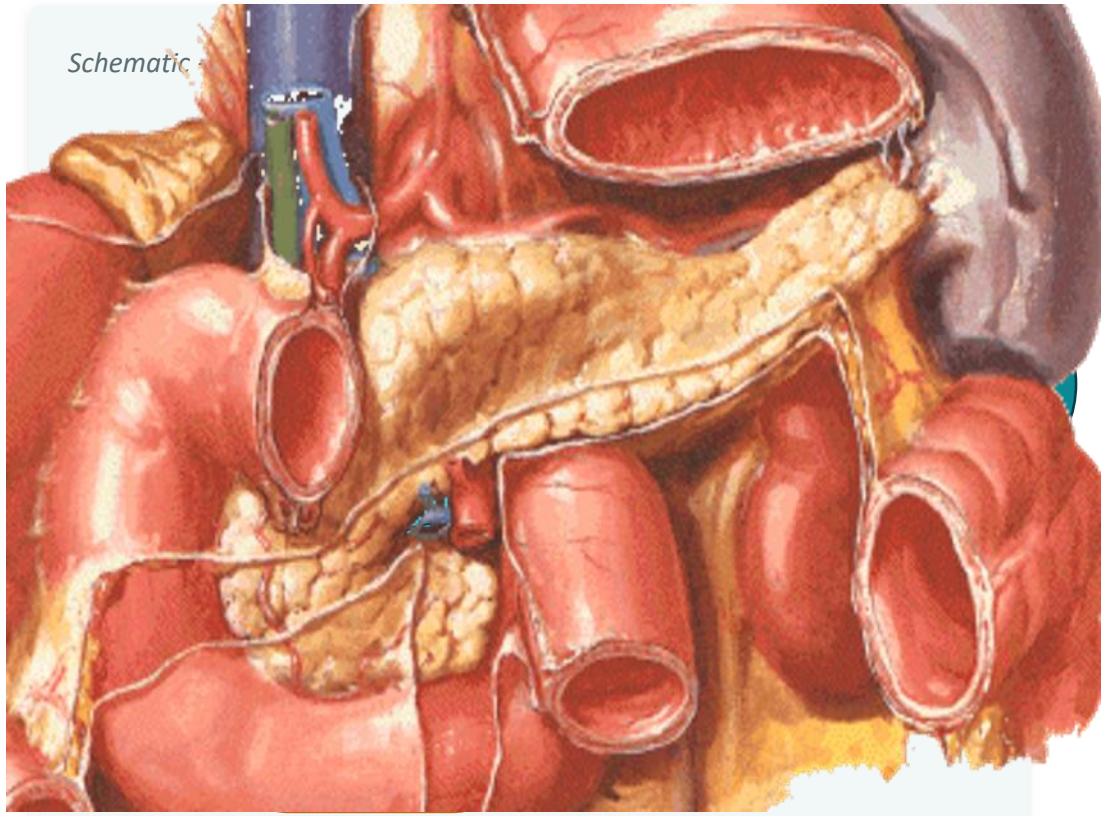
Lies transversely across L1–L2; secondary retroperitoneal position explains why fluid/inflammation tracks into the retroperitoneum and paracolic gutters.

Four parts

Head (with uncinate process) nestles in the duodenal C-loop; neck overlies the SMV/portal vein confluence; body crosses the aorta & L1; tail reaches the splenic hilum.

Why it matters clinically

Head-region disease can obstruct the CBD (jaundice); neck disease threatens the portal/SMV confluence (splenic vein thrombosis); tail disease can injure the spleen.



L1–L2 vertebral level, retroperitoneal

Relations, Ductal System & Vessels

Ductal anatomy
Main pancreatic duct (of Wirsung) joins the CBD at the ampulla of Vater; accessory duct (of Santorini) drains separately — obstruction here raises intraductal pressure and triggers autodigestion.

Arterial supply
Splenic artery (body & tail) and superior/inferior pancreaticoduodenal arcades from the gastroduodenal & SMA (head) — explains bleeding risk in necrotising pancreatitis (pseudoaneurysm of splenic/GDA vessels).

Venous drainage
Splenic vein runs directly behind the pancreas and joins the SMV to form the portal vein — inflammation/necrosis here causes splenic vein thrombosis and sinistral (left-sided) portal hypertension.

Key posterior relations
Aorta, IVC, left renal vein, and the transverse mesocolon attach at the anterior surface — explains why severe pancreatitis causes retroperitoneal collections and paralytic ileus.

CAUSES

Etiology — “I GET SMASHED”

- **I** Idiopathic
- **G** **Gallstones (most common)**
- **E** **Ethanol (alcohol — 2nd most common)**
- **T** Trauma
- **S** Steroids
- **M** Mumps & other viruses (CMV, coxsackie)
- **A** Autoimmune (IgG4-related)
- **S** Scorpion sting
- **H** Hypercalcaemia / Hyperlipidaemia
- **E** ERCP (post-procedure)
- **D** Drugs (azathioprine, thiazides, valproate)

MECHANISM

Pathogenesis

1. Initiating insult

Ductal obstruction / alcohol toxicity / ischaemia injures the acinar cell.

2. Premature enzyme activation

Trypsinogen is prematurely converted to trypsin within the acinar cell.

3. Autodigestion

Trypsin activates other proenzymes (elastase, phospholipase A2) causing local tissue digestion.

4. Cytokine release

Acinar injury releases TNF-α, IL-1, IL-6 driving local & systemic inflammation.

5. SIRS / organ failure

Systemic cytokine spill-over produces SIRS, capillary leak, and in severe cases multi-organ failure.

Net result: local pancreatic/peripancreatic necrosis plus a systemic inflammatory response — the basis for both local and systemic complications discussed later.

CLASSIFICATION

Revised Atlanta Classification (2012)

Interstitial Oedematous Pancreatitis

Diffuse (or occasionally localised) enlargement of the pancreas due to inflammatory oedema; homogeneous or mildly heterogeneous enhancement on CECT; peripancreatic fat shows inflammatory changes.

Necrotising Pancreatitis

Necrosis of pancreatic parenchyma and/or peripancreatic tissue (~5–10% of cases); non-enhancing areas on CECT; higher risk of infection and organ failure.

Severity grading

Grade	Organ failure	Local complications
Mild	None	None
Moderately severe	Transient (<48h)	May be present
Severe	Persistent (>48h), single/multi-organ	Usually present

PRESENTATION

Clinical Features

Pain
Sudden, severe epigastric pain radiating to the back; classically relieved by leaning forward.

Vomiting
Nausea and repeated vomiting that does not relieve the pain.

Systemic signs
Fever, tachycardia, hypotension; epigastric tenderness with guarding in severe disease.

Hallmark Signs of Haemorrhagic Pancreatitis

Grey Turner's sign

Bluish-brown discolouration of the flanks from retroperitoneal haemorrhage tracking laterally.

Cullen's sign

Periumbilical bluish discolouration from haemoperitoneum tracking via the falciform ligament.

Fox's sign

Ecchymosis over the inguinal ligament.

These signs indicate severe necrotising, haemorrhagic pancreatitis and carry a poor prognosis.

Differential Diagnosis

Condition	Distinguishing feature
Perforated peptic ulcer	Sudden onset, board-like rigidity; free air under diaphragm on erect X-ray
Acute cholecystitis	RUQ pain, Murphy's sign positive; USG shows gallbladder wall thickening
Mesenteric ischaemia	Pain out of proportion to exam, often in atrial fibrillation/atherosclerosis
Ruptured AAA / aortic dissection	Pulsatile abdominal mass, hypotension, back pain in an older patient
Inferior wall MI	ECG changes, cardiac enzyme rise; may mimic epigastric pain
Intestinal obstruction	Colicky pain, distension, absolute constipation; air-fluid levels on X-ray

Laboratory Workup

Serum lipase

More sensitive & specific than amylase; rises within 4–8h, stays elevated 8–14 days.

Serum amylase

Rises within hours, normalises in 3–5 days; level does not correlate with severity.

LFTs

ALT > 150 IU/L within 48h suggests gallstone aetiology (~95% specific).

Calcium & lipid profile

Hypocalcaemia is a poor prognostic sign; triglycerides > 1000 mg/dL suggest hyperlipidaemic aetiology.

CRP

CRP > 150 mg/L at 48h correlates with severe/necrotising disease.

CBC, renal function, ABG

Haematocrit, urea/creatinine and blood gases feed directly into severity scores.

Imaging

Transabdominal Ultrasound
First-line — screens for gallstones/biliary dilatation; pancreas often obscured by bowel gas.

Contrast-Enhanced CT (CECT)
Best after 72–96h if diagnosis unclear or severe disease suspected — early scans may underestimate necrosis.

MRCP / EUS
Used to detect choledocholithiasis or occult microlithiasis, especially in “idiopathic” cases.

Balthazar CT Severity Index (CTSI)

Grade	CT finding	Pts
A	Normal pancreas	0
B	Pancreatic enlargement	1
C	+ Peripancreatic inflammation	2
D	+ Single fluid collection	3
E	≥ 2 collections / gas	4

Necrosis score added: 0% = 0, ≤30% = 2, 30–50% = 4, >50% = 6 pts. Total CTSI (0–10) predicts complication rate & mortality.

Severity Scoring Systems

System	Timing	Key parameters	Notes
Ranson's criteria	At admission + 48h	Age, WBC, glucose, LDH, AST (admission); fall in Hct, rise in urea, Ca, PaO_2, base deficit, fluid sequestration (48h)	≥3 predicts severe disease; cannot be completed before 48h
APACHE II	Any time, repeatable	Physiological variables + age + chronic health	Score ≥8 = severe; can be recalculated daily
BISAP	Within 24h	BUN>25, Impaired mental status, SIRS, Age>60, Pleural effusion	Simple bedside score; ≥3 predicts mortality
CTSI (Balthazar)	On CECT (72–96h)	Inflammation grade + % necrosis	Correlates with local complications

Local Complications

Acute peripancreatic fluid collection (APFC)
Occurs within 4 weeks in interstitial oedematous pancreatitis; no defined wall; usually resolves spontaneously.

Pancreatic pseudocyst
Encapsulated fluid collection with a well-defined wall, forming after >4 weeks; lacks solid debris.

Acute necrotic collection (ANC) / Walled-off necrosis (WON)
Contains solid necrotic debris; becomes “walled-off” after 4 weeks. Prone to infection.

Infected necrosis
Suspected with clinical deterioration, gas on CT, or positive FNA culture — the leading indication for intervention.

Systemic Complications

ARDS

Due to circulating phospholipase A2 & cytokines damaging alveolar-capillary membranes.

Acute kidney injury

From hypovolaemia, hypotension and systemic inflammation.

Shock

Third-space fluid loss into the retroperitoneum → hypovolaemic/distributive shock.

Hypocalcaemia

From saponification of peripancreatic fat and reduced PTH response — a poor prognostic marker.

Hyperglycaemia

From islet cell (β-cell) injury and stress response.

DIC

Systemic activation of the coagulation cascade in severe disease.

Initial Resuscitation (First 24–48h)

Aggressive IV fluid resuscitation
Goal-directed crystalloid (e.g. Ringer's lactate); the single most important early intervention to prevent necrosis.

Analgesia
Adequate pain control, typically opioids, titrated to effect.

Monitor & supportive care
Urine output, vitals, oxygen saturation; ICU/HDU for severe disease; correct electrolytes.

Withhold oral intake initially
NPO in the acute phase, then resume oral feeding as tolerated once pain & inflammation settle.

Serial severity assessment
Repeat scoring (Ranson at 48h, daily SIRS/organ function) to identify evolving severe disease early.

Nutritional Support

Mild pancreatitis

Oral feeding can start as soon as pain improves and nausea settles, generally within 24–72h — a low-fat, soft diet is well tolerated.

Severe pancreatitis

Early enteral nutrition (nasojejunal or nasogastric) within 24–72h is preferred over total parenteral nutrition — it maintains gut mucosal barrier and reduces infectious complications.

Key point: Total parenteral nutrition is reserved for patients who cannot tolerate enteral feeding (e.g. ileus, intolerance) — enteral feeding is otherwise preferred whenever the gut works.

Antibiotics & Role of ERCP

Antibiotics

- Not recommended routinely / prophylactically in sterile necrosis
- Indicated for confirmed infected necrosis, cholangitis, or extrapancreatic infection (e.g. pneumonia, UTI, line sepsis)
- Choose agents with good pancreatic penetration — carbapenems, quinolones + metronidazole

ERCP in Gallstone Pancreatitis

- Urgent ERCP (within 24–72h) indicated only if concomitant cholangitis or persistent biliary obstruction
- Not routinely required in mild gallstone pancreatitis without obstruction/cholangitis
- MRCP/EUS can clarify ductal stones non-invasively before committing to ERCP

Necrotising Pancreatitis: The Step-Up Approach

1. Delay

Wherever possible, delay intervention beyond 4 weeks to allow the collection to "wall off," improving safety of drainage/debridement.

2. Percutaneous / endoscopic drainage

Image-guided percutaneous catheter drainage or endoscopic transgastric drainage as the first step.

3. Minimally invasive necrosectomy

If drainage alone is insufficient — video-assisted retroperitoneal debridement (VARD) or endoscopic necrosectomy.

4. Open necrosectomy

Reserved for failure of minimally invasive steps or when not feasible — highest morbidity, used as last resort.

Principle: "escalate only as needed" — start with the least invasive effective option and step up only if the patient fails to improve.

Indications for Intervention

Infected pancreatic/peripancreatic necrosis
The leading indication — suspected with clinical sepsis, gas on CT, or positive FNA/blood culture.

Symptomatic sterile necrosis / WON
Persistent pain, gastric outlet or biliary obstruction, or a growing collection despite conservative care.

Abdominal compartment syndrome
Intra-abdominal pressure >20 mmHg with new organ dysfunction — may need decompressive laparotomy.

Disconnected pancreatic duct / bleeding
Pseudoaneurysm rupture (angioembolisation first-line) or persistent duct leak with recurrent collections.

Symptomatic pseudocyst
Large (>6 cm), symptomatic, or complicated pseudocysts not resolving after 6 weeks of observation.

Procedures for Pancreatitis Complications

Procedure	Used for	Notes
Image-guided percutaneous catheter drainage (PCD)	Infected/symptomatic collections — first step	Simple, low morbidity; may be definitive in ~35–50% of cases
Endoscopic transluminal drainage ± necrosectomy	WON accessible via stomach/duodenum	Cystogastrostomy with stents/LAMS; repeat endoscopic necrosectomy as needed
Video-assisted retroperitoneal debridement (VARD)	Necrosis after failed PCD	Minimally invasive; via a percutaneous drain tract
Open necrosectomy ± lavage	Failure of minimally invasive steps, diffuse necrosis	Highest morbidity/mortality; reserved as last resort
Cystogastrostomy / cystojejunostomy (surgical)	Mature symptomatic pseudocyst	Internal drainage once the cyst wall is well-formed (>6 weeks)

Cholecystectomy in Gallstone Pancreatitis

Mild gallstone pancreatitis

Laparoscopic cholecystectomy during the same admission, once clinically improving — reduces risk of recurrent pancreatitis/biliary events.

Severe / necrotising pancreatitis

Cholecystectomy is delayed until the acute inflammatory process and any collections have resolved (often several weeks later), to reduce operative risk.

Interval ERCP with sphincterotomy is an alternative to cholecystectomy in patients unfit for surgery.

Broader Pancreatic Surgery: A Quick Overview

Whipple's procedure (pancreaticoduodenectomy)
Resection of the pancreatic head, duodenum, distal stomach/pylorus, CBD & gallbladder — for periampullary/pancreatic head tumours, and occasionally for refractory groove pancreatitis.

Distal pancreatectomy ± splenectomy
Removal of the body/tail — used for tumours or trauma to the distal pancreas; spleen-preserving where feasible.

Puestow procedure (lateral pancreaticojejunostomy)
Longitudinal duct decompression for chronic pancreatitis with a dilated main duct ("chain-of-lakes") — relieves ductal hypertension.

Frey procedure
Local resection of the pancreatic head core combined with lateral pancreaticojejunostomy — for chronic pancreatitis with an inflammatory head mass plus ductal dilatation.

OUTCOMES

Prognosis & Follow-up

~1–3%

mortality in mild interstitial pancreatitis

~15–30%

mortality in infected necrotising pancreatitis

~30%

risk of recurrence if the underlying cause is not corrected

Address the cause
Cholecystectomy for gallstone pancreatitis; strict alcohol cessation counselling; correct hyperlipidaemia/hypercalcaemia.

Monitor for chronic sequelae
Recurrent attacks can progress to chronic pancreatitis with exocrine/endocrine insufficiency (steatorrhoea, diabetes).

Long-term follow-up
Reassess persistent collections/pseudocysts, nutritional status, and glycaemic control at follow-up visits.

SUMMARY

Key Takeaways

1. Diagnosis needs 2 of 3: typical pain, enzymes >3x normal, or characteristic imaging.
2. Gallstones and alcohol are the two leading causes worldwide.
3. Aggressive early fluid resuscitation in the first 24–48h is the cornerstone of management.
4. The revised Atlanta classification and scoring systems (Ranson, APACHE II, BISAP, CTSI) guide severity assessment.
5. Infected necrosis is the key indication for intervention — follow the step-up approach and delay drainage/necrosectomy past 4 weeks where possible.
6. Cholecystectomy timing depends on severity: same-admission for mild disease, delayed for severe/necrotising disease.

Thank you — Questions?