FINAL MBBS • SURGERY

Chronic Pancreatitis

& Pancreatic Pseudocyst

Etiology, pathogenesis, clinical features, and a complete guide to medical, endoscopic and surgical management

A structured, exam-oriented review

ROADMAP

Learning Objectives

Define & classify

Define chronic pancreatitis and outline the TIGAR-O aetiological classification.

Pathogenesis

Explain how recurrent injury leads to fibrosis, ductal change and organ dysfunction.

Clinical triad

Recognise pain, steatorrhoea and diabetes as the hallmark presentation.

Investigate & manage

Select appropriate imaging/functional tests and step through medical–endoscopic–surgical care.

Surgical options

Compare drainage vs resection procedures and know when each is indicated.

Pseudocyst

Define, diagnose and manage a pancreatic pseudocyst, distinguishing it from other cystic lesions.

OVERVIEW

Chronic Pancreatitis: Definition & Epidemiology

Definition

Chronic pancreatitis is a progressive, irreversible inflammatory disease of the pancreas characterised by continuing fibrosis, destruction of exocrine and endocrine parenchyma, and permanent structural change — leading over time to pain and both exocrine and endocrine insufficiency.

Key distinction from acute pancreatitis

- ✦ Structural and functional changes are permanent, not reversible
- ✦ Recurrent acute attacks may punctuate the chronic course
- ✦ Pain, steatorrhoea and diabetes reflect cumulative parenchymal loss

5–12 per 100,000 annual incidence worldwide

70–80% of cases in the West attributable to alcohol

M > F male predominance, typically 4th–5th decade

↑ Risk long-standing disease increases pancreatic cancer risk

CAUSES

Etiology — the TIGAR-O Classification

T	**Toxic–Metabolic**	Alcohol (commonest), smoking, hypercalcaemia, hyperlipidaemia, chronic renal failure, drugs, toxins
I	**Idiopathic**	Early- and late-onset idiopathic chronic pancreatitis; tropical pancreatitis
G	**Genetic**	PRSS1 (hereditary pancreatitis), CFTR, SPINK1 mutations
A	**Autoimmune**	IgG4-related (Type 1) and idiopathic duct-centric (Type 2) autoimmune pancreatitis
R	**Recurrent/severe acute pancreatitis**	Post-necrotic scarring, recurrent acute attacks, vascular disease, post-irradiation
O	**Obstructive**	Pancreas divisum, duct strictures, ampullary stenosis, tumours obstructing the duct

MECHANISM

Pathogenesis

Necrosis–fibrosis sequence
Repeated bouts of acute injury (e.g. alcohol-related) cause necrosis, which heals with progressive peri- and intralobular fibrosis over time.

SAPE hypothesis (Sentinel Acute Pancreatitis Event)
An initial acute attack activates pancreatic stellate cells; recurrent insults perpetuate stellate cell activation and drive relentless fibrogenesis.

Oxidative stress / toxic-metabolic injury
Alcohol metabolites and oxidative stress directly injure acinar cells and promote ductal protein plugging and stone formation.

Ductal obstruction theory
Protein plugs/calculi obstruct small ducts, raising intraductal pressure, causing upstream acinar atrophy and fibrosis.

Pathological & Ductal Changes

Parenchymal fibrosis & atrophy

Progressive loss of acinar tissue replaced by dense fibrous stroma, reducing exocrine and endocrine reserve.

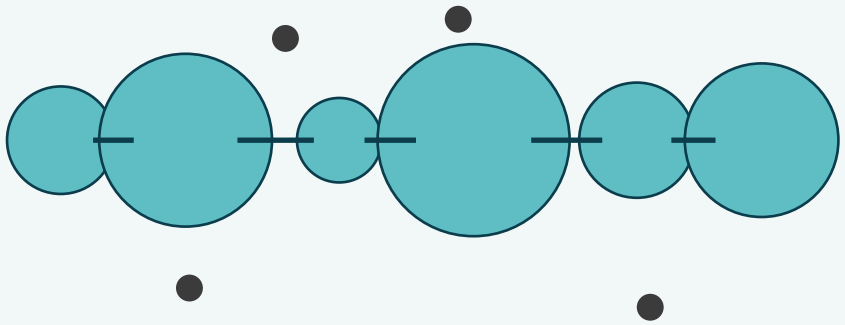
Ductal dilatation & stricturing

Alternating strictures and dilatations produce the classic 'chain of lakes' appearance of the main pancreatic duct.

Intraductal calculi

Protein plugs calcify into ductal stones, a hallmark radiological and pathological finding, especially in alcohol-related disease.

Schematic — “chain of lakes” duct



Irregular duct dilatation & stricturing with intraductal calculi (calcifications)

PRESENTATION

Clinical Features — The Classic Triad

Abdominal pain

Recurrent or constant epigastric pain radiating to the back; may lessen as burnt-out fibrosis reduces gland function (‘burnout’ phenomenon).

Steatorrhoea (exocrine insufficiency)

Bulky, foul-smelling, floating stools from fat malabsorption; occurs once >90% of exocrine function is lost. Weight loss is common.

Diabetes mellitus (endocrine insufficiency)

'Type 3c' diabetes from loss of islet cells; often brittle, with both insulin and glucagon deficiency.

Note: the full triad appears late — pain is usually the earliest and most common presenting feature; steatorrhoea and diabetes reflect advanced parenchymal destruction.

Complications of Chronic Pancreatitis

Pseudocyst formation

Ductal disruption leads to a peripancreatic fluid collection walled by fibrous/granulation tissue (discussed in detail later).

Splenic/portal vein thrombosis

Peripancreatic inflammation causes splenic vein thrombosis — sinistral portal hypertension with gastric varices.

Biliary & duodenal obstruction

Fibrosis of the pancreatic head compresses the intrapancreatic CBD (obstructive jaundice) or duodenum (gastric outlet obstruction).

Pancreatic ascites / pleural effusion

Ductal disruption with leakage of pancreatic fluid into the peritoneal or pleural cavity; ascitic fluid amylase is markedly elevated.

Pancreatic cancer

Long-standing chronic pancreatitis (especially hereditary forms) carries a significantly increased lifetime risk of pancreatic adenocarcinoma.

Imaging

Plain abdominal X-ray
May show speckled pancreatic calcification in the epigastrium — a simple, classic (though insensitive) finding.

Contrast-enhanced CT
Best initial cross-sectional test — shows gland atrophy, calcification, ductal dilatation and complications (pseudocyst, vascular).

MRCP
Non-invasive ductal imaging of choice — delineates strictures, dilatation and the 'chain of lakes' pattern without ionising contrast risk.

Endoscopic ultrasound (EUS)
Most sensitive test for early parenchymal and ductal change; also allows FNA and guided interventions.

Functional Tests

Direct (invasive) tests

Secretin/CCK stimulation test — the most sensitive & specific test of exocrine function; measures pancreatic bicarbonate/enzyme output via duodenal aspirate after hormonal stimulation. Gold standard but invasive and not widely available.

Indirect (non-invasive) tests

- ✦ Faecal elastase-1 — simple stool test; low levels (<200 µg/g) indicate exocrine insufficiency; widely used first-line test
- ✦ Faecal fat estimation (72-hour) — quantifies steatorrhoea (>7 g fat/day is abnormal) but cumbersome for patients
- ✦ Serum trypsinogen — low levels support advanced exocrine insufficiency

Imaging Classification — Cambridge Grading

Grade	Main duct	Side branches	Other features
Normal	Normal calibre	Normal	None
Equivocal	Normal	<3 abnormal	None
Mild	Normal	≥3 abnormal	None
Moderate	Abnormal calibre	≥3 abnormal	None
Marked (severe)	Abnormal + one or more of:	≥3 abnormal	Large cavity, obstruction, filling defects, calculi, gland destruction

Note: the M-ANNHEIM classification is an alternative comprehensive system combining aetiology, clinical stage and severity — mentioned here for completeness; Cambridge grading remains the most widely used imaging-based system.

Medical Management

Lifestyle modification
Complete alcohol abstinence and smoking cessation — the single most important step to slow disease progression and reduce pain.

Analgesia
Stepwise (WHO ladder) approach from NSAIDs to opioids; coeliac plexus block/neurolysis for refractory pain in selected patients.

Pancreatic enzyme replacement therapy (PERT)
Oral pancreatic enzymes with meals treat steatorrhoea and aid nutrition; often combined with proton pump inhibitors to protect enzymes from acid degradation.

Diabetes management
Insulin is often required (Type 3c/pancreatogenic diabetes); patients are prone to hypoglycaemia due to concurrent glucagon deficiency.

Nutritional support
Fat-soluble vitamin (A, D, E, K) supplementation and dietary counselling; small, frequent, low-fat meals are better tolerated.

Endoscopic Management

Endoscopic sphincterotomy ± stone extraction
For ductal calculi, especially combined with extracorporeal shockwave lithotripsy (ESWL) to fragment large stones first.

Pancreatic duct stenting
Relieves strictures and improves ductal drainage, providing symptomatic pain relief in selected patients.

EUS-guided coeliac plexus block
Endoscopic ultrasound-guided neurolysis for pain control, particularly useful when surgery is not desired or feasible.

Endoscopic therapy is best suited to ductal stones/strictures without a significantly dilated duct or a large inflammatory mass — surgery is generally more durable when the duct is markedly dilated.

Indications for Surgery

Intractable pain
Pain refractory to maximal medical and endoscopic therapy is the most common indication for surgery.

Suspected malignancy
An inflammatory head mass that cannot confidently be distinguished from cancer warrants resection.

Complications needing intervention
Symptomatic pseudocyst, biliary or duodenal obstruction, pancreatic fistula, or vascular complications.

Ductal anatomy favouring surgery
A markedly dilated main pancreatic duct (>7 mm, 'chain of lakes') responds well to surgical drainage procedures.

Duct-Drainage Procedures

Puestow procedure (Partington-Rochelle modification)

A longitudinal lateral pancreaticojejunostomy: the main pancreatic duct is opened along its length and anastomosed side-to-side to a Roux loop of jejunum, draining the entire 'chain of lakes'. Best suited to a dilated duct (≥7 mm) without a dominant head mass. The original Puestow-Gillesby also resected the tail with splenectomy; the modern Partington-Rochelle modification preserves the spleen and tail.

Key advantage & limitation

Advantage: preserves pancreatic parenchyma and avoids resection-related endocrine/exocrine loss, with good short-term pain relief. Limitation: does not address a dominant inflammatory head mass — in that setting a hybrid resection-drainage procedure (Frey/Beger, next slide) gives superior and more durable pain relief.

Hybrid Resection-Drainage Procedures

Frey procedure

Local excision (coring) of the pancreatic head combined with a longitudinal lateral pancreaticojejunostomy. Preserves the duodenum, CBD and stomach while decompressing the duct and removing the diseased head core. Widely favoured for its lower morbidity.

Beger procedure

Duodenum-preserving pancreatic head resection: the pancreatic head is resected (leaving a thin rim along the duodenum and CBD), followed by a Roux-en-Y pancreaticojejunostomy to both the resection cavity and the distal duct. More extensive resection than Frey.

Resection Procedures

Procedure	What is removed	When used
Pancreaticoduodenectomy (Whipple's)	Pancreatic head, duodenum, distal stomach/pylorus, CBD, gallbladder	Head-dominant disease where malignancy cannot be excluded
Distal pancreatectomy ± splenectomy	Body/tail of the pancreas	Disease localised to the body/tail (e.g. post-traumatic stricture)
Total pancreatectomy ± islet autotransplantation	Entire pancreas; islet cells harvested and reinfused into the liver via the portal vein	Diffuse small-duct disease with intractable pain refractory to all other options

Islet autotransplantation aims to preserve some endocrine function despite total gland removal, reducing the severity of resulting diabetes — an increasingly used adjunct in specialist centres.

OUTCOMES

Long-Term Outcomes of Chronic Pancreatitis

~50%

achieve good long-term pain relief after surgical drainage/resection

~4%

lifetime risk of pancreatic cancer with long-standing disease (much higher in hereditary pancreatitis)

↓ Life expectancy

from disease complications and continued alcohol/tobacco use

Progressive course
Exocrine and endocrine insufficiency typically worsen over years even after pain improves ('burnout').

Continued surveillance
Regular nutritional, glycaemic, and (in hereditary/high-risk disease) pancreatic cancer surveillance is recommended.

PART TWO

Pancreatic Pseudocyst

Definition, diagnosis and management of the commonest cystic complication of pancreatitis

DEFINITION

Pancreatic Pseudocyst: Definition & Pathogenesis

Definition (Revised Atlanta Classification)

An encapsulated collection of fluid with a well-defined inflammatory wall, usually outside the pancreas, with minimal or no necrotic debris. It typically takes more than 4 weeks from the onset of pancreatitis to form a mature wall.

Following acute pancreatitis
An acute peripancreatic fluid collection persists beyond 4 weeks and develops a fibrous wall, without significant necrotic content.

Following chronic pancreatitis / duct disruption
A side-branch or main duct disruption leaks pancreatic secretions that collect and become walled off — often communicates with the main duct.

Following trauma
Blunt abdominal trauma disrupting the pancreatic duct is an important cause, especially in younger patients.

PRESENTATION

Clinical Features & Diagnosis

Persistent abdominal pain / epigastric mass
Ongoing or recurrent pain after an attack of pancreatitis, sometimes with a palpable epigastric mass.

Early satiety, nausea, weight loss
From gastric or duodenal compression by a large cyst.

Complications at presentation
Rarely, presents with infection (fever), rupture, haemorrhage (pseudoaneurysm into the cyst), or biliary obstruction.

Diagnostic work-up

- Contrast-enhanced CT — defines size, wall, contents and relation to surrounding structures
- MRCP — assesses communication with the main pancreatic duct, important for planning drainage
- EUS — characterises the cyst wall/contents and allows fine-needle aspiration to exclude a neoplastic cyst
- Cyst fluid analysis — high amylase, low CEA typically favours a pseudocyst over a mucinous neoplasm

Differential Diagnosis of Pancreatic Cystic Lesions

Lesion	Typical features	Malignant potential
Pseudocyst	History of pancreatitis/trauma; high fluid amylase, low CEA; unilocular, no solid component	None (not a neoplasm)
Serous cystadenoma	Microcystic 'honeycomb' pattern, central scar; low CEA	Essentially benign
Mucinous cystic neoplasm	Usually body/tail, thick wall, may have septations; high CEA in fluid	Premalignant — often resected
Intraductal papillary mucinous neoplasm (IPMN)	Communicates with the pancreatic duct; main-duct type has higher malignant risk	Premalignant, variable risk

Key teaching point: a history of preceding pancreatitis plus high amylase/low CEA fluid strongly favours pseudocyst; without that history, a cystic neoplasm must be actively excluded before assuming 'pseudocyst'.

Natural History & Indications for Intervention

Natural history

A significant proportion of pseudocysts, especially those <6 cm and of recent onset, resolve spontaneously with conservative management and observation — intervention is not automatic just because a pseudocyst is present.

Indications for intervention

Size & duration
Large (>6 cm) and persistent beyond 6 weeks — traditionally cited as reducing the chance of spontaneous resolution and raising complication risk.

Symptomatic cysts
Persistent pain, gastric outlet or biliary obstruction due to mass effect.

Complicated cysts
Infection, haemorrhage (pseudoaneurysm), or rupture require urgent intervention regardless of size.

Drainage Options

Approach	Technique	Notes
Percutaneous drainage	Image-guided catheter placement into the cyst	Useful for infected or rapidly enlarging cysts; risk of persistent pancreatico-cutaneous fistula if duct communication present
Endoscopic drainage	EUS-guided cystogastrostomy/cystoduodenostomy with stents (plastic pigtail or lumen-apposing metal stents)	Now first-line for mature, endoscopically accessible cysts adjacent to the stomach/duodenum — avoids surgery in most patients
Surgical drainage	Open or laparoscopic cystogastrostomy, cystoduodenostomy, or Roux-en-Y cystojejunostomy	Reserved for large, multiloculated, or surgically inaccessible cysts, or when endoscopic drainage fails/is unavailable

Principle: wait for a mature (≥4 week old), well-defined cyst wall before drainage wherever possible — this reduces complications and recurrence, mirroring the 'delay' principle used in necrotising pancreatitis.

SUMMARY

Key Takeaways

1. Chronic pancreatitis is progressive and irreversible — alcohol/tobacco (TIGAR-O 'toxic-metabolic') is the leading cause worldwide.

2. The classic triad — pain, steatorrhoea, diabetes — reflects cumulative parenchymal destruction and usually appears late.

3. MRCP/CT show the 'chain of lakes' duct; faecal elastase is the simplest screening test for exocrine insufficiency.

4. Management is stepwise: lifestyle change and analgesia → endoscopic ductal therapy → surgery for intractable pain or complications.

5. Choose drainage (Puestow) for a dilated duct, a hybrid procedure (Frey/Beger) for a dominant head mass, and resection (Whipple/distal/total) when malignancy can't be excluded or disease is diffuse.

6. A pancreatic pseudocyst is a mature, non-neoplastic fluid collection (>4 weeks) — many resolve spontaneously; intervene for symptoms, complications, or persistent large size, with endoscopic drainage now first-line.

Thank you — Questions?