

# Pancreatitis

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# Acute pancreatitis

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- An acute condition presenting with abdominal pain, a threefold or greater rise in the serum levels of the pancreatic enzymes amylase or lipase, and/ or characteristic findings of pancreatic inflammation on contrast- enhanced CT.

# mechanism

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- Premature activation of pancreatic enzyme within the pancreas, leading to a process of autodigestion.
- Anything that injures the acinar cell and impairs the secretion of zymogen granules or damages the duct epithelium and thus delays enzymatic secretion, can trigger acute pancreatitis.
- Once cellular injury has been initiated, the inflammatory process can lead to pancreatic edema, hemorrhage and, eventually, necrosis

As inflammatory mediators are released into the circulation, systemic complications can arise, such as

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- Hemodynamic instability
- Bacteremia (due to translocation of gut flora)
- Acute respiratory distress syndrome and pleural effusions
- Gastrointestinal hemorrhage
- Renal failure
- Disseminated intravascular coagulation (DIC)

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- Acute pancreatitis may be categorized as mild (interstitial edematous pancreatitis) or severe (necrotizing pancreatitis).
  - The former is characterized by interstitial edema of the gland and minimal organ dysfunction.
  - The majority of patients will have a mild attack of pancreatitis, the mortality from which is around 1%.

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- Severe acute pancreatitis is seen in 5–10% of patients, and is characterized by pancreatic necrosis, a severe systemic inflammatory response and often multi-organ failure
  - In those who have a severe attack of pancreatitis, the mortality varies from 20 to 50%.

# Early phase

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- It is characterized by a systemic inflammatory response syndrome (SIRS) which – if severe – can lead to transient or persistent organ failure (deemed persistent if it lasts for over 48 hours).
- About one-third of **deaths** occur in the early phase of the attack, **from multiple organ failure**.
- It usually lasts a week

# late phase

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- Typically, in those who suffer a severe attack and can run from weeks to months.
- It is characterized by persistent systemic signs of inflammation, and/or local complications, particularly fluid collections and peripancreatic sepsis.
- **Deaths** occurring after the first week of onset are often due to **septic complications**.

## Acute pancreatitis

- The possible causes
- The two major causes of acute pancreatitis are biliary calculi, which occur in 50–70% of patients, and alcohol abuse, which accounts for 25% of cases

## Possible causes of acute pancreatitis

- Gallstones
- Alcoholism
- Post ERCP
- Abdominal trauma
- Following biliary, upper gastrointestinal or cardiothoracic surgery
- Ampullary tumour
- Drugs (corticosteroids, azathioprine, asparaginase, valproic acid, thiazides, oestrogens)
- Hyperparathyroidism
- Hypercalcaemia
- Pancreas divisum
- Autoimmune pancreatitis
- Hereditary pancreatitis
- Viral infections (mumps, coxsackie B)
- Malnutrition
- Scorpion bite
- Idiopathic

## Clinical presentation

- Pain is the cardinal symptom.
- Nausea, repeated vomiting and retching are usually marked accompaniments.
- On examination, look for ( vital signs and signs of dehydration , jaundice , abdominal distention and guarding , gray turner sign and Cullen's sign , signs of pleural effusion and pulmonary edema , CNS exam looking for confusion secondary to hypoxemia)

## *Investigations*

- Typically, the diagnosis is made on the basis of the **clinical presentation** and an **elevated serum amylase level**
- A serum amylase level three times above normal is indicative of the disease..
- Normal level up to 140 U/L
- A normal serum amylase level does not exclude acute pancreatitis particularly if the patient has presented a few days later.
- Other ( CBC, KFT LFT , LDH ,ABGS ,Ca , Blood sugar )

Investigations in acute pancreatitis should be aimed at answering three questions:

- Is a diagnosis of acute pancreatitis correct?
- How severe is the attack?
- What is the aetiology?

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- **Lipase level : if the amylase level is normal**
  - more sensitive and specific test than amylase.
  - If there is doubt, and other causes of acute abdomen have to be excluded, contrast-enhanced CT is probably the best single imaging investigation.
  - Causes of elevated amylase ( Renal failure , IO, perforated DU )

# ASSESSMENT OF SEVERITY

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Regardless of the system used, persisting organ failure indicates a severe attack.

A serum C-reactive protein level  $>150$  mg/L at 48 hours after the onset of symptoms is also an indicator of severity.

Patients with a body mass index over 30 are at higher risk of developing complications.

There is no correlation between amylase level and severity of the attack

# Atlanta classification of acute pancreatitis

- **Mild acute pancreatitis:**
  - no organ failure
  - no local or systemic complications.
- **Moderately severe acute pancreatitis:**
  - organ failure that resolves within 48 hours (transient organ failure); and/or
  - local or systemic complications without persistent organ failure.
- **Severe acute pancreatitis:**
  - persistent organ failure (>48 hours);
  - single organ failure;
  - multiple organ failure

# Ranson's criteria

## Ranson's prognostic signs of pancreatitis

### Criteria for acute pancreatitis not due to gallstones

At admission	During the initial 48 h
Age >55 y	Hematocrit fall >10 points
WBC >16,000/mm <sup>3</sup>	BUN elevation >5 mg/dL
Blood glucose >200 mg/dL	Serum calcium <8 mg/dL
Serum LDH >350 IU/L	Arterial PO <sub>2</sub> <60 mmHg
Serum AST >250 U/dL	Base deficit >4 mEq/L
	Estimated fluid sequestration >6 L

### Criteria for acute gallstone pancreatitis

At admission	During the initial 48 h
Age >70 y	Hematocrit fall >10 points
WBC >18,000/mm <sup>3</sup>	BUN elevation >2 mg/dL
Blood glucose >220 mg/dL	Serum calcium <8 mg/dL
Serum LDH >400 IU/L	Base deficit >5 mEq/L
Serum AST >250 U/dL	Estimated fluid sequestration >4 L

# Glasgow score

A score more than 3  
indicate sever attack

Glasgow score

**Within 48 hours**

Age >55 years

White blood cell count  $>15 \times 10^9/L$

Blood glucose  $>10 \text{ mmol/L}$  (no history of diabetes)

LDH  $> 600 \text{ units/L}$

Serum urea  $>16 \text{ mmol/L}$  (no response to intravenous fluids)

Arterial oxygen saturation ( $\text{PaO}_2$ )  $<8 \text{ kPa}$  (60 mmHg)

Serum calcium  $<2.0 \text{ mmol/L}$

Serum albumin  $<32 \text{ g/L}$

# IMAGING

- **Abdominal x ray + Plain erect chest:**
- Not diagnostic of acute pancreatitis but are useful in the differential diagnosis.
- Non-specific findings:
  - Generalized or local ileus (sentinel loop)
- Occasionally, calcified gallstones or pancreatic calcification may be seen.
- A chest radiograph may show a pleural effusion and, in severe cases, acute respiratory distress syndrome.



Calcified  
GBS



Pancreatic  
calcification

# Sentinel loop



# Ultrasound

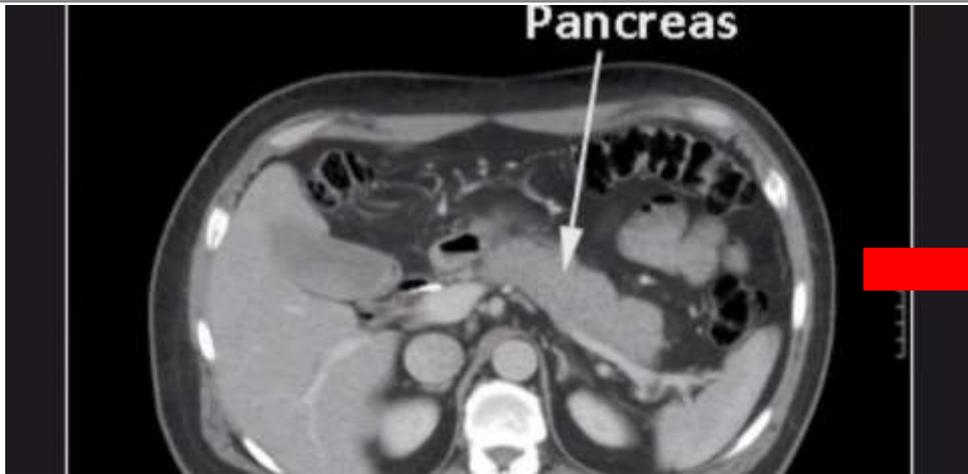
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- Ultrasound does not establish a diagnosis of acute pancreatitis.
- The swollen pancreas may be seen.
- Ultrasonography should be performed within 24 hours in all patients to detect gallstones as a potential cause, rule out acute cholecystitis as a differential diagnosis and determine whether the common bile duct is dilated.

# CT scan

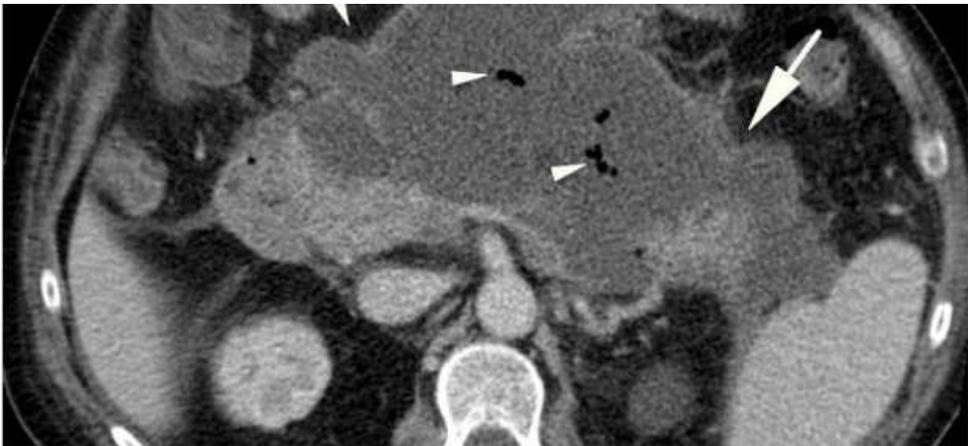
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- CT is not necessary for all patients
  - If there is diagnostic uncertainty.
    - In patients with severe acute pancreatitis, to distinguish interstitial from necrotizing pancreatitis .
  - In patients with organ failure, signs of sepsis or progressive clinical deterioration.
- When a localized complication is suspected, such as fluid collection, pseudocyst or a pseudoaneurysm



→ Normal  
pancrease

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→ Infected pancreatic  
necrosis

# MRCP & ERCP

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- MRCP can help in detecting stones in the common bile duct and directly assessing the pancreatic parenchyma
- ERCP allows the identification and removal of stones in the common bile duct in gallstone pancreatitis.

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multiple round filling defects  
within the gallbladder



round filling defect  
within dilated distal  
common bile duct

# Management

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- **Mild attack**
- Noninvasive approach
- Intravenous fluid administration
- brief period of fasting may be sensible in a patient who is nauseated and in pain
- Antibiotics are not indicated.
- Analgesia
- Antiemetic
- Supplement oxygen

**TABLE 68.4** Early management of severe acute pancreatitis.

Admission to HDU/ICU

Analgesia

Aggressive fluid rehydration

Supplemental oxygen

Invasive monitoring of vital signs, central venous pressure, urine output, blood gases

Frequent monitoring of haematological and biochemical parameters (including liver and renal function, clotting, serum calcium, blood glucose)

Nasogastric drainage (only initially)

Antibiotics if cholangitis suspected;

CT scan essential if organ failure, clinical deterioration or signs of sepsis develop

ERCP within 72 hours for severe gallstone pancreatitis or signs of cholangitis

Supportive therapy for organ failure if it develops (inotropes, ventilatory support, haemofiltration, etc.)

If nutritional support is required, consider enteral (nasogastric) feeding

# antibiotic ??

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- Prophylactic antibiotic isn't recommended
- Antibiotic in pancreatitis only if there is infected pancreatic necrosis or other documented infection ( UTI , chest infection , cholangitis , acute cholecystitis ...etc )

**TABLE 68.5** Complications of acute pancreatitis.

<b>Systemic</b>	<b>Local</b>
<i>(More common in the first week)</i>	<i>(Usually develop after the first week)</i>
Cardiovascular	Acute fluid collection
Shock	Sterile pancreatic necrosis
Arrhythmias	Infected pancreatic necrosis
Pulmonary	Pancreatic abscess
ARDS	Pseudocyst
Renal failure	Pancreatic ascites
Haematological	Pleural effusion
DIC	Portal/splenic vein thrombosis
Metabolic	Pseudoaneurysm
Hypocalcaemia	
Hyperglycaemia	
Hyperlipidaemia	
Gastrointestinal	
Ileus	
Neurological	
Visual disturbances	
Confusion, irritability	
Encephalopathy	
Miscellaneous	
Subcutaneous fat necrosis	
Arthralgia	

# STERILE AND INFECTED PANCREATIC NECROSIS

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- diffuse or focal area of non-viable parenchyma.
- This can be identified by an absence of parenchymal enhancement on CT with intravenous contrast.
- It associated with lysis of peripancreatic fat

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- *acute necrotic collection* (ANC).
  - This is typically an intra or extra pancreatic collection containing fluid and necrotic material, with no definable wall.
  - Gradually, over a period of over 4 weeks, this may develop a well-defined inflammatory capsule, and evolve into what is termed *walled-off necrosis* (WON).

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- Collections associated with necrotizing pancreatitis are sterile to begin with but often become subsequently infected, probably due to translocation of gut bacteria.
  - Infected necrosis is associated with a mortality rate of up to 50%.
  - Sterile necrotic material should not be drained or interfered with.

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- if the patient shows signs of sepsis, then one should determine whether the collection is infected.
  - CT scan with needle insertion ( avoid passage through the hollow viscus ).
  - The aspirate should be sent for microbiological assessment, and appropriate antibiotic therapy should be commenced as per the sensitivity report.+ drain insertion
  - If the sepsis worsens despite this, then a pancreatic necrosectomy should be considered

# ACUTE PERIPANCREATIC FLUID COLLECTION (APFC)

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- It has no encapsulating wall and is confined within normal fascial planes.
- The fluid is sterile, and most such collections resolve.
- No intervention is necessary unless a large collection causes symptoms or pressure effects, in which case it can be percutaneously aspirated under ultrasound or CT guidance
- early course of Mild pancreatitis without evidence of necrosis

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- a fluid collection appearing in the first 4 weeks after the onset of pancreatitis is an acute fluid collection; after 4 weeks, it becomes an acute pseudocyst
  - The most common complaint is recurrent or persistent upper abdominal pain.
  - **CT** is the radiographic study of choice for initial evaluation of pancreatic pseudocysts.
  - Other ( MRCP , ERCP) evaluation of ductal anatomy

# Pancreatic pseudocyst

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- Pseudocysts **smaller than 4 cm** usually resolve spontaneously.
  - Complication : infection , bleeding ( erosion most commonly to splenic artery ) , obstruction , rupture , enteric fistula .
  - **Nonoperative.** If the pseudocyst is new, asymptomatic, and without complications, the patient can be followed with serial CT scans or US.
  - Drainage ( internal or external ) or excision.

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- Nutritional support: Naso-jejunal or TPN
  - Cholecystectomy is indicated on the index admission as a definitive management of gallstone pancreatitis
  - Delay has resulted in the occurrence of a second attack, which may be more severe.
  - In patients with severe gallstone pancreatitis, cholecystectomy should be performed when general and local conditions permit

# Chronic pancreatitis

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- Chronic pancreatitis is a progressive inflammatory disease in which there is irreversible destruction of pancreatic tissue.
- Its clinical course is characterized by severe pain and, in the later stages, exocrine and endocrine pancreatic insufficiency.
- In the early stages of its evolution, it is frequently complicated by attacks of acute pancreatitis, which are responsible for the recurrent pain that may be the only clinical symptom

# Causes

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- High alcohol consumption is the most frequent cause of chronic pancreatitis, accounting for 60–70% of cases.
- pancreatic duct obstruction
- pancreas divisum and annular pancreas, if associated with papillary stenosis
- Hereditary pancreatitis, CF, infantile malnutrition
- metabolic (hypercalcemia, hypertriglyceridemia, hypercholesterolemia, hyperparathyroidism),

# Clinical features

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- Pain
- Nausea and vomiting
- Weight loss
- Impact on the social life
- Steatorrhea
- DM

# Investigation

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- Only in the early stages of the disease will there be a rise in serum amylase, usually amylase and lipase are normal
- Pancreatic calcifications may be seen on abdominal X-ray
- CT or MRI scan will show the outline of the gland, the main area of damage and the possibilities for surgical correction

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- An MRCP will identify the presence of biliary obstruction and the state of the pancreatic duct
  - ERCP is the most accurate way of elucidating the anatomy of the duct and, in conjunction with the whole organ morphology, can help to determine the type of operation required, if operative intervention is indicated

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- **Pancreatic secretin stimulation tests**
  - highly sensitive (90% to 100%) and specific (>90%) test for the diagnosis of chronic pancreatitis.
  - **A 72-hour fecal collection for estimation of daily fecal fat**
  - **Pancreatic endocrine function**

# Treatment

## Medical treatment of chronic pancreatitis

### Treat the addiction

- Help the patient to stop alcohol consumption and tobacco smoking
- Involve a dependency counsellor or a psychologist

### Alleviate abdominal pain

- Eliminate obstructive factors (duodenum, bile duct, pancreatic duct)
- Escalate analgesia in a stepwise fashion
- Refer to a pain management specialist
- For intractable pain, consider CT/EUS-guided coeliac axis block

### Nutritional and pharmacological measures

- Diet: low in fat and high in protein and carbohydrates
- Pancreatic enzyme supplementation with meals
- Correct malabsorption of the fat-soluble vitamins and vitamin B12
- Micronutrient therapy with methionine, vitamins C & E, selenium (may reduce pain and slow disease progression)
- Steroids (only in autoimmune pancreatitis, for relief of symptoms)
- Medium-chain triglycerides in patients with severe fat malabsorption (they are directly absorbed by the small intestine without the need for digestion)
- Reducing gastric secretions may help

### Treat diabetes mellitus

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- Endoscopic, radiological or surgical interventions are indicated mainly to relieve obstruction of the pancreatic duct, bile duct or the duodenum, or in dealing with complications (e.g. pseudocyst, abscess, fistula, ascites or variceal haemorrhage).