

# Lecture 3: pancreatitis

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# Objectives

- Recognize the predisposing factors of pancreatitis.
- Describe the different types of pancreatitis.
- Understand the pathogenesis of acute and chronic pancreatitis

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# Pancreatitis

**Definition :** Inflammation of the **pancreas**.

- The clinical manifestations can range in severity from a mild, self-limited disease to a life threatening acute inflammatory process.
- The duration of the disease can range from a transient attack (reversible) to an irreversible loss of function.
- It could be **acute** or **chronic** .

# Acute pancreatitis

**Definition :** a group of **reversible** lesions characterized by inflammation of the pancreas.

**Table 16-1** Etiologic Factors in Acute Pancreatitis

## Metabolic

Alcoholism\* ←  
Hyperlipoproteinemia  
Hypercalcemia  
Drugs (e.g., azathioprine)

## Genetic

Mutations in the cationic trypsinogen (*PRSS1*) and trypsin inhibitor (*SPINK1*) genes

## Mechanical

Gallstones\* ←  
Trauma  
Iatrogenic injury  
    Perioperative injury  
    Endoscopic procedures with dye injection

## Vascular

Shock  
Atheroembolism  
Polyarteritis nodosa

## Infectious

Mumps  
Coxsackievirus

\*Most common causes in the United States.

**N.B.:** the smaller gallstone is the more dangerous to cause pancreatitis



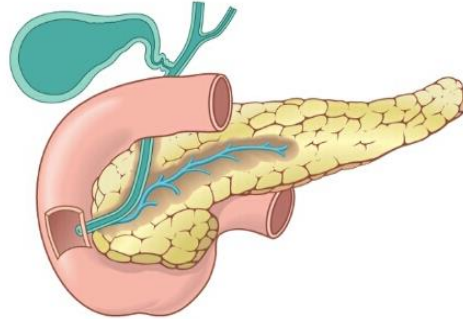
# Pathogenesis :

## Auto digestion of pancreatic substance by inappropriate activated pancreatic enzymes.



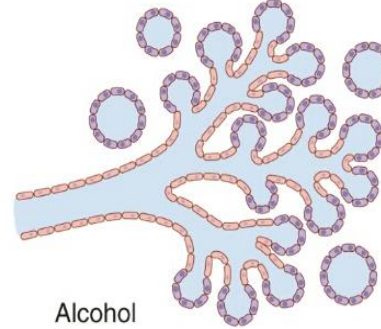
### CAUSES:

#### DUCT OBSTRUCTION



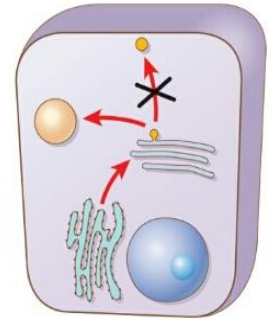
- Cholelithiasis
- Ampullary obstruction
- Chronic alcoholism
- Ductal concretions

#### ACINAR CELL INJURY



- Alcohol
- Drugs
- Trauma
- Ischemia
- Viruses

#### DEFECTIVE INTRACELLULAR TRANSPORT



- Metabolic injury (experimental)
- Alcohol
- Duct obstruction

### MECHANISMS:

Interstitial edema

Impaired blood flow

Ischemia

Release of intracellular proenzymes and lysosomal hydrolases

Activation of enzymes (intra- or extracellular)

Delivery of proenzymes to lysosomal compartment

Intracellular activation of enzymes

Acinar cell injury

ACTIVATED ENZYMES

### LESIONS:

Interstitial inflammation and edema

+

Proteolysis (proteases)

+

Fat necrosis (lipase, phospholipase)

+

Hemorrhage (elastase)

ACUTE PANCREATITIS

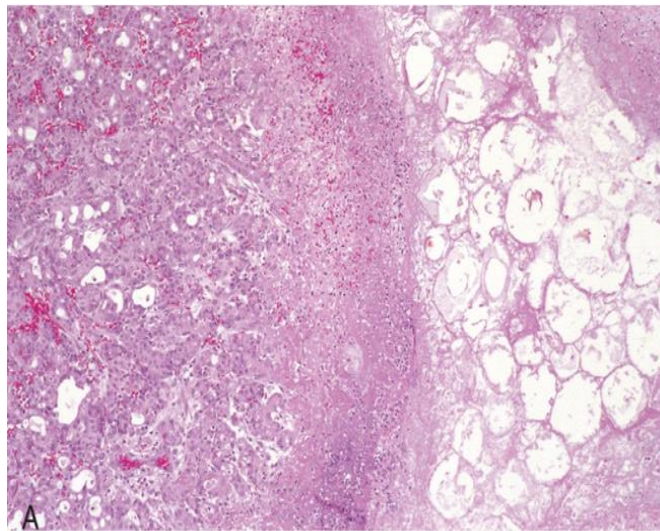
## Morphology :

The morphology of acute pancreatitis ranges from inflammation and edema to severe extensive necrosis and haemorrhage .

- **The basic alterations are :**

1. Microvascular leakage causing **edema** .
2. Necrosis of **fat** (by lipases which destructs fat cells).
3. An **acute** inflammatory reaction .
4. Proteolytic **destruction** of pancreatic **parenchyma**.
5. Destruction of blood vessels with subsequent interstitial **hemorrhage** .

- The alteration depend on the **Etiology**. fat necrosis is the **most common**.
- The released fatty acids combine with calcium to form **insoluble** salts that precipitate.



Region of fat necrosis (right) and focal pancreatic parenchymal necrosis (center).



Dark areas of hemorrhage in the pancreatic substance and a focal area of pale fat necrosis in the peripancreatic fat (upper left).



## Clinical Features :

- **Abdominal pain** (Major symptoms).
- Full-blown acute pancreatitis is a **medical emergency**, usually have the sudden onset of an “**acute abdomen**”, The pain is constant and intense and is often referred to the **upper back**.

## Laboratory findings :

- elevation of **serum amylase** levels during the first 24 hours(earlier )
- rising **serum lipase** level within 72 to 96 hours (more specific) .

## Management and Prognosis :

The key to the management is "**resting**" the pancreas by total restriction of food and fluids and by supportive therapy. Nothing by mouth “**NBM or NPO = nil per os**”

- Most patients recover fully.
- About 5% die from shock during the first week of illness.
- **Acute respiratory distress syndrome** and **acute renal failure** are fatal complications.
- In surviving patients, sequelae\* include:
  - I. Sterile pancreatic **abscess**
  - II. Pancreatic **pseudocyst**.

\* A chronic condition that is a complication of an acute condition that begins during that acute condition.

# Chronic pancreatitis

**Definition** : inflammation of the pancreas with destruction of **exocrine** parenchyma, **fibrosis**, and **irreversible** impairment in pancreatic function .

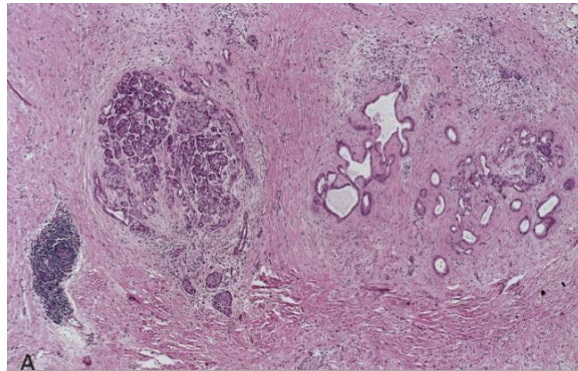
- in the **late stages**, also destruction of **endocrine** parenchyma .

**Causes** : **Similar** to the acute form, patients are usually **middle-aged** males .

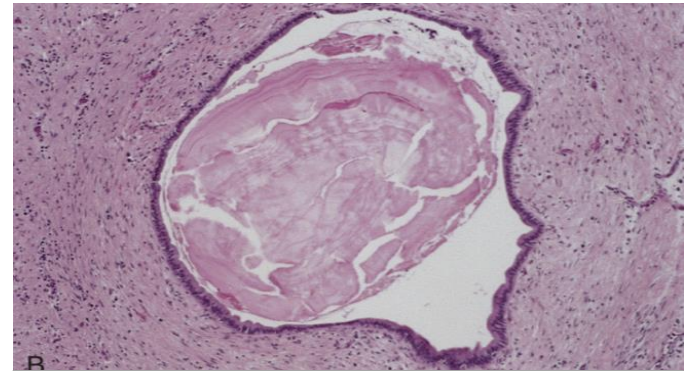
- **Idiopathic** chronic pancreatitis .
- Tropical pancreatitis\* .

**Morphology** :

❖ **Grossly**: gland is **hard**, with extremely dilated ducts and visible **calcification** .



- 1- fibrosis
- 2- atrophy
- 3- residual islets (**left**) and ducts (**right**),
- 4- chronic inflammatory cells
- 5- acinar tissue.



**Higher magnification**

- 1- Dilated ducts
- 2- inspissated (thick) eosinophilic concretions

\*Disease seen in africa and asia. It has been attributed to malnutrition .







## Clinical Features :

- Silent or repeated attacks of **abdominal pain** Or persistent abdominal
- **back pain.**
- Attacks may be precipitated by **alcohol** abuse, **overeating** ,or the use of opiates and other drugs.
- There may be mild **fever** .
- mild-to-moderate elevations of serum **amylase**.
- CT scan and MRI shows **Calcifications** within the pancreas .

## Complications :

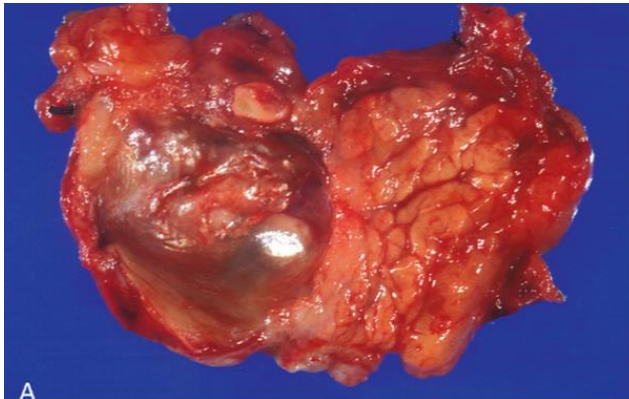
- Chronic **malabsorption** .
- Severe pancreatic exocrine insufficiency .
- **Diabetes mellitus** (due to destruction of islets of Langerhans) .
- Severe chronic pain .
- **Pancreatic pseudocysts** .

# Pseudocysts of pancreas

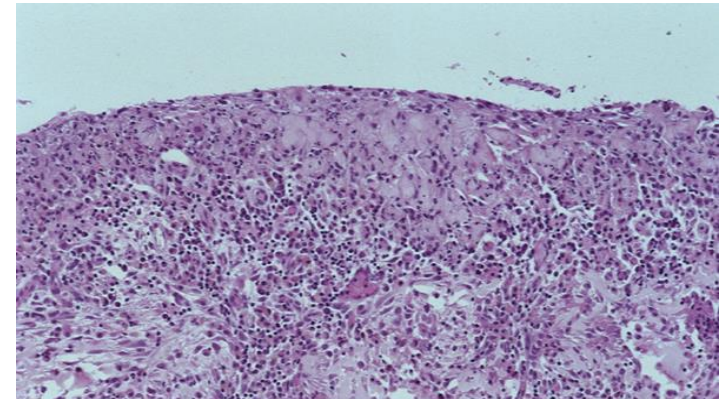
**Definition :** localized collections of **necrotic-hemorrhagic** material rich in pancreatic **enzymes**.

- Lack an **epithelial lining** "pseudo" .
- They account for majority of cysts in the pancreas.
- Usually arise after an episode of **acute** pancreatitis, or of **chronic** alcoholic pancreatitis.

**Morphology :**



Poorly defined cyst with a necrotic brownish wall.



The cyst lacks a true epithelial lining and instead is lined by fibrin and granulation tissue, with typical changes of chronic inflammation.

**Complications :** They can produce abdominal pain and predispose to **intraperitoneal hemorrhage** or **peritonitis**.



# Summary from Robbins



## SUMMARY

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### Pancreatitis

- *Acute pancreatitis* is characterized by inflammation and reversible parenchymal damage that ranges from focal edema and fat necrosis to widespread parenchymal necrosis and hemorrhage; the clinical presentation varies widely, from mild abdominal pain to rapidly fatal vascular collapse.
- *Chronic pancreatitis* is characterized by irreversible parenchymal damage and scar formation; clinical presentations include chronic malabsorption (due to pancreatic exocrine insufficiency) and diabetes mellitus (due to islet cell loss).
- Both entities share similar pathogenic mechanisms, and indeed recurrent acute pancreatitis can result in chronic pancreatitis. *Ductal obstruction* and *long-term alcohol abuse* are the most common causes in both forms. Inappropriate activation of pancreatic digestive enzymes (due to mutations in genes encoding trypsinogen or trypsin inhibitors) and primary acinar injury (due to toxins, infections, ischemia, or trauma) also cause pancreatitis.

