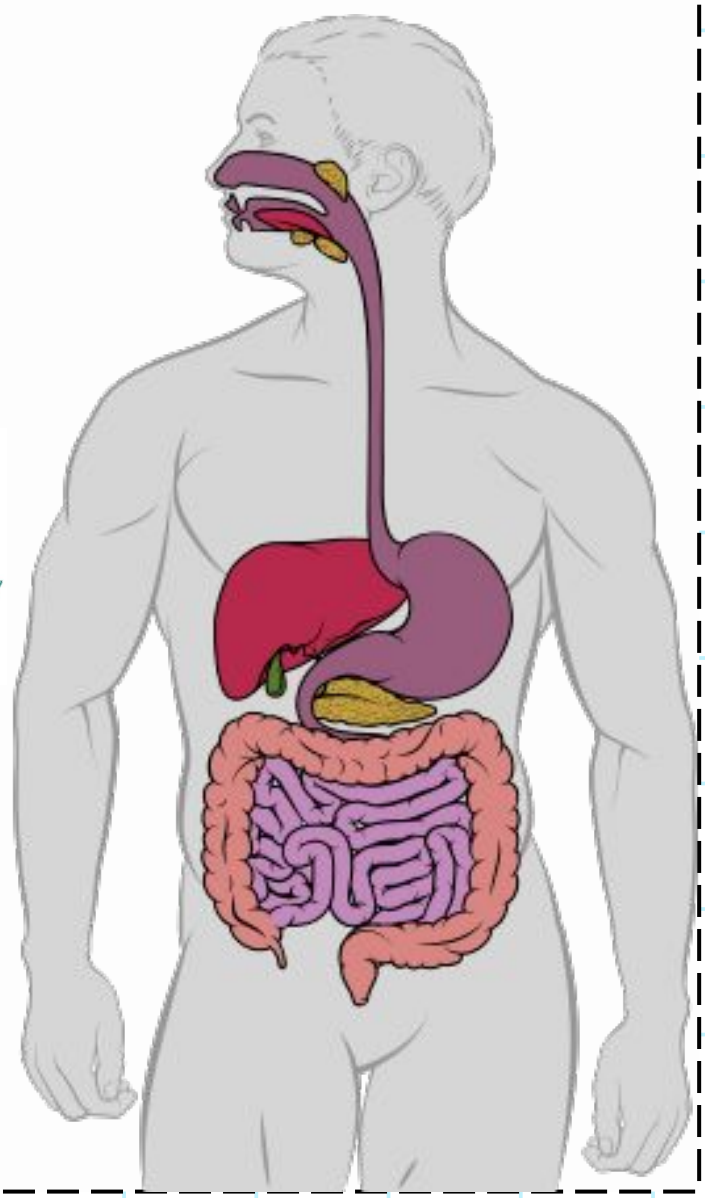
Acute & Chronic pancreatitis



GNT BLOCK

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Editing File

Color index :
Main text (black)
Female Slides (Pink)
Male Slides (Blue)
Important (Red)
Dr's note (Green)
Extra Info (Grey)

Male Slides

OBJECTIVES

- Describe the pathology of acute and chronic pancreatitis.
- Understand the pathogenesis of acute and chronic pancreatitis
- Describe the clinical features and possible complications of acute and chronic pancreatitis.

THIS LECTURE WAS PRESENTED BY DR.WAJD ALTHAGAFI & DR.AHMED ALHUMAIDI

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Introduction

The pancreas is really two organ packaged into one

Endocrine portion (10% of pancreas)

- Islets of Langerhans : secrete insulin, glucagon, and somatostatin (hormones) .
- The most significant disorders of the endocrine pancreas are **diabetes mellitus** and neoplasms.

Exocrine portion (90% of pancreas)

- Makes up the bulk of this organ is a major source of enzymes that are essential for digestion.
- Acinar cells, the ductules and ducts that convey their secretions to the duodenum.

Pancreatitis

Definition	Clinical manifestation	Types
It encompasses a group of disorders characterized by inflammation of the pancreas.	Can range in severity from: Mild self-limited disease → to a life-threatening Idiopathic acute inflammatory process.	**1- Acute Pancreatitis** The gland can return to normal if the underlying cause of pancreatitis is removed ,reversible . **2- Chronic Pancreatitis** Irreversible loss of exocrine pancreatic parenchyma.

Acute Pancreatitis

Definition

Reversible pancreatic parenchymal injury associated with inflammation

Epidemiology

Relatively common, with an annual incidence rate in **Western** countries of 10 to 20 cases per 100,000 people.

Biliary tract diseases and alcoholism account for approximately 80% of cases in Western countries.

Gallstones are present in 35% to 60% of cases of acute pancreatitis, and about 5% of patients with gallstones develop pancreatitis.

The male-to-female ratio is :
- 1 : 3 in the group with biliary tract diseases (females)
- 6 : 1 in those with alcoholism (men) .

Etiologic Factors

All in females slides except blue in males

Metabolic	- **Alcoholism** - Hyperlipoproteinemia - Hypercalcemia - Medications (85 drugs e.g, azathioprine, sulfonamides)
Genetic	- Germline Mutations in the cationic trypsinogen (**PRSSI**) , and trypsin inhibitor (**SPINKI**) genes (recurrent severe acute pancreatitis often beginning in childhood)
Mechanical	- Gallstones & Obstruction of the pancreatic duct - Trauma. - Iatrogenic injury. - Perioperative injury. - Endoscopic procedures with dye injection.
Vascular	- Shock - Atheroembolism - Vasculitis:Polyarteritis nodosa - Thrombosis
Infections	- Mumps - Coxsackievirus - Parasitic : Ascaris

Acute Pancreatitis

Pathogenesis

Three pathways can incite the initial enzyme activation that may lead to acute pancreatitis:

Acute pancreatitis appears to be caused by autodigestion of the pancreas by inappropriately activated pancreatic enzymes.hyperactivation of trypsin, also hyperactivation of many other digestive enzymes that require trypsin cleavage for their activation and may leading to tissue injury and inflammation. The pancreas is normally protected from autodigestion by synthesis of pancreatic enzymes in the acinar cells in the proenzymes form.

Female Slides

Pancreatic Duct Obstruction	- Blocks ductal low ➔ ↑ intraductal pressure & accumulation of enzyme-rich interstitial fluid ➔lipase (secreted in an active form)➔ local fat necrosis. - Injured tissues + periacinar myofibroblasts + leukocytes release pro-inflammatory cytokines ➔ promote local inflammation and interstitial edema. - Edema ➔ compromises local blood low ➔ vascular insufficiency & ischemic injury to acinar cells.
Primary Acinar Cell Injury	- This pathogenic pathway comes into play in acute pancreatitis caused by ischemia, viral infections, drugs & direct trauma to the pancreas.
Defective Intracellular Transport of Proenzymes Within Acinar Cells	- Proenzyme activation ➔ lysosomal rupture ➔ local release of activated enzymes. In normal acinar cells : After synthesis in the ER, digestive proenzymes goes to zymogen granules & hydrolytic enzymes goes to lysosomes and are transported in discrete pathways. In animal models: pancreatic proenzymes & lysosomal hydrolases become packaged together ➔ proenzyme activation ➔lysosomal rupture ➔ local release of activated enzymes. The role of this mechanism in human acute pancreatitis is not clear.

Alcohol consumption may causes pancreatitis by several mechanisms :

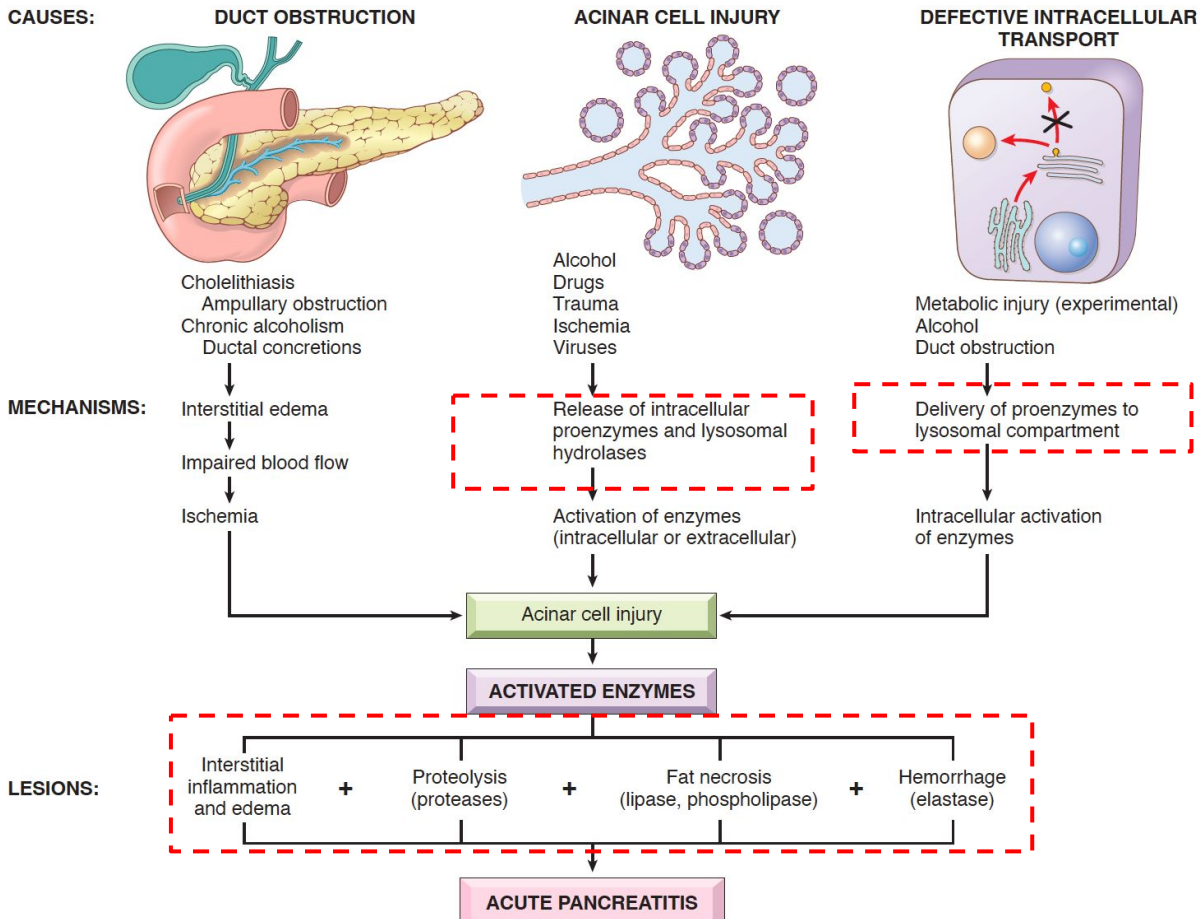
Female Slides

01 Alcohol transiently increases pancreatic exocrine secretion & contraction of the sphincter of Oddi.

02 Alcohol has direct toxic effects on acinar cells.

03 Chronic alcohol ingestion ➔ secretion of protein-rich pancreatic fluid ➔ deposition of inspissated protein plugs➔ obstruction of small pancreatic ducts.

Acute Pancreatitis



tant

Three pathways can incite the initial enzyme activation that may lead to acute pancreatitis:

1. **Pancreatic duct obstruction :** Impaction of a gallstone or biliary sludge, or extrinsic compression of the ductal system by a mass blocks ductal flow, increases intraductal pressure, and allows accumulation of an enzyme- rich interstitial fluid. Since lipase is secreted in an active form, local fat necrosis may result. Injured tissues, periacinar myofibroblasts, and leukocytes then release proinflammatory cytokines that promote local inflammation and interstitial edema through a leaky microvasculature. Edema further compromises local blood flow, causing vascular insufficiency and ischemic injury to acinar cells.
2. **Primary acinar cell injury :** This pathogenic mechanism comes into play in acute pancreatitis caused by ischemia, viral infections (e.g., mumps), drugs, and direct trauma to the pancreas.
3. **Defective intracellular transport of proenzymes within acinar cells :** In normal acinar cells, digestive enzymes intended for zymogen granules (and eventually extracellular release) and hydrolytic enzymes destined for lysosomes are transported in discrete pathways after synthesis in the endoplasmic reticulum.

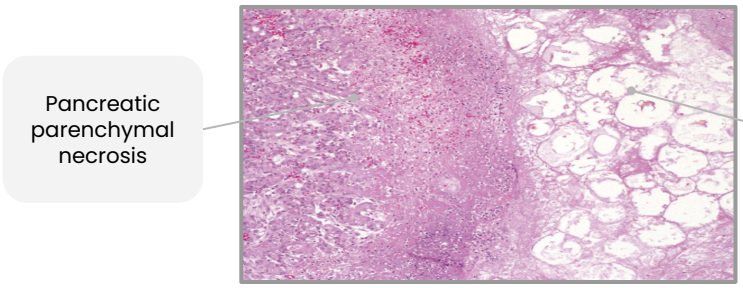
Acute Pancreatitis

Histologically

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

The basic alterations

1. Microvascular leakage causing **edema**
2. **Fat necrosis** by lipolytic enzymes (**lipases**)
3. Acute inflammation (neutrophils)
4. Proteolytic destruction of pancreatic parenchyma by proteases
5. Destruction of blood vessels and subsequent **interstitial hemorrhage** due to activation of elastases



Acute necrotizing pancreatitis (sever form)

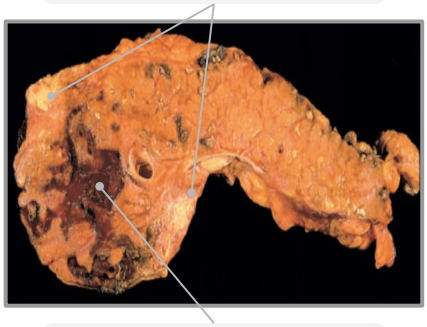
- Necrosis of pancreatic acinar & ductal tissues as well as the islets of langerhans
- Vascular damage ➔ hemorrhage into the parenchyma of the pancreas
- Fat necrosis can occur in extra-pancreatic fat, including the omentum & pancreatitis bowel mesentery, and even outside the abdominal cavity (e.g, in (sever form)subcutaneous fat)
- Peritoneum usually contains a serous, slightly turbid, brown-tinged fluid with globules of fat (derived from enzymatically digested adipose tissue).

Hemorrhagic Pancreatitis

Extensive parenchymal necrosis and diffuse hemorrhage leading to shock. (most severe)

Grossly

- Red-black hemorrhage interspersed with foci of yellow-white, chalky fat necrosis
- Fat necrosis results from enzymatic destruction of fat cells. The released fatty acids combine with calcium to form insoluble salts that precipitate in situ (appear as foci of yellow-white, chalky material)
- Foci of fat necrosis may also be found in extra-pancreatic collections of fat, including the omentum and bowel mesentery, and even outside the abdominal cavity



Acute Pancreatitis

Clinical Features

01

Abdominal pain : varies in severity from mild & uncomfortable to sever & incapacitating

02

Characteristically, the pain is constant, intense & often is referred to upper back

03

Full-blown acute pancre constitutes a medical emergency "acute abdomen": sudden onset of abdominal pain, abdominal guarding/rigidity & absence of bowel sounds

04

80% of cases are mild & self limiting; remaining 20% develop severe disease ➔ shock, hemorrhage & chronic pancreatitis

05

It must be differentiated from : Ruptured acute appendicitis, perforated peptic ulcer, acute cholecystitis with rupture & occlusion of mesenteric Clinical vessels with infarction of bowel

06

Systemic release of digestive enzymes + activation of inflammatory response ➔ leukocytes, disseminated intravascular coagulation (DIC), acute respiratory distress syndrome (due to alveolar capillary injury) & diffuse fat necrosis

07

↑Microvascular permeability → hypovolemia → peripheral vascular collapse (shock) → acute tubular necrosis in kidney → acute renal failure eventually multi organ failure

08

disseminated intravascular coagulation may be related to early intravascular consumption of coagulation factors secondary to circulating pancreatic enzymes, particularly trypsin Pleural effusion due to transdiaphragmatic lymphatic blockage

09

Severe pancreatic damage leads to systemic inflammatory response syndrome (SIRS) and affects multiple organs including lungs, the severe form of which is acute respiratory distress syndrome (ARDS)

Clinical Note

The deep location of the pancreas sometimes gives rise to problems of diagnosis for the following reasons:

- Because the pancreas lies behind the stomach and transverse colon, disease of the gland can be confused with that of the stomach or transverse colon.
- Inflammation of the pancreas can spread to the peritoneum forming the posterior wall of the lesser sac. This in turn can lead to adhesions and the closing off of the lesser sac to form a pseudocyst.

Acute Pancreatitis

Diagnosis

1. **Laboratory findings include** markedly elevated serum **amylase** during the first 24 hours, followed (within 72–96hours) by rising serum lipase levels.
2. **Hypocalcemia** (due to precipitation of calcium in areas of fat necrosis) If persistent, it is a poor prognostic sign.
3. CT scan or MRI will show enlarged & inflamed pancreas.

Management	**Supportive therapy:** such as maintaining BP & alleviating pain, "Resting" the pancreas by total restriction of food & fluids, and Treat the primary cause.
Prognosis	• Most patients will eventually recover • 5% can die from shock during the first week of illness
Complication	• Some may develop acute respiratory distress syndrome +/- and acute renal failure are fatal complications. • Survivors may develop: - Sterile or infected pancreatic "abscesses" - Pancreatic pseudocysts

Clinical Note

Because pancreas is responsible for fat digestion , many fat soluble vitamins will be decreased thus patient could present with :

FAT-SOLUBLE VITAMIN DEFICIENCY	
DEFICIENCY	**SYMPTOMS**
VITAMIN A	• Night blindness (nyctalopia) • Dry, scaly skin (xerosis cutis) • Bitot spots (keratin debris; foamy appearance on conjunctiva) • Corneal degeneration (keratomalacia) • Immunosuppression
VITAMIN D	• Rickets (children), osteomalacia (adults) • Hypocalcemia → hypocalcemic tetany
VITAMIN E	• Hemolytic anemia • Acanthocytosis • Muscle weakness • Demyelination of posterior columns (position/vibration sense loss, ataxia)
VITAMIN K	• Hemorrhage (esp. neonatal) • Prolonged PT and aPTT with normal bleeding time

Pancreatic pseudocysts

A common sequela of acute pancreatitis (alcoholic pancreatitis)

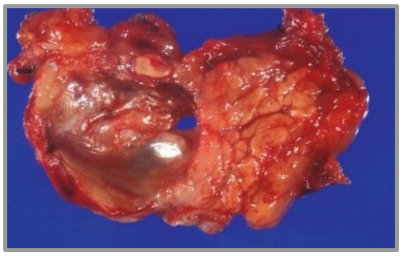
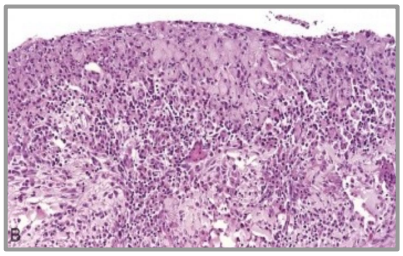
Pseudocysts are localized collections of liquefied areas of necrotic tissue-hemorrhagic material rich in pancreatic enzymes, the areas become walled off by fibrous tissue to form a cystic space, **lacking an epithelial lining** (hence the prefix "pseudo").

They account for majority of cysts in the pancreas, approximately accounting for 75% of all pancreatic cysts.

Traumatic injury to the abdomen can also give rise to pseudocysts.

Morphology Pseudocyst of Pancreas

- While many pseudocysts spontaneously resolve, they may become secondarily infected, and larger pseudocysts may compress or even perforate into adjacent structures.
- They can produce abdominal pain and predispose to intraperitoneal hemorrhage or peritonitis.

Grossly	Histologically
A poorly defined cyst with a necrotic brownish wall, usually solitary and can range in size from 2 to 30 cm in diameter. 	lacks an epithelial lining and instead is lined by fibrin and granulation tissue, with typical changes of chronic inflammation 

Deep Focus Question

Which of the following is NOT a systemic complication of acute pancreatitis?

A. Pleural effusion
B. Kidney failure
C. DIC
D. Shock
E. Phlegmon

Answer: E

Deep Focus Question

Which of the following is NOT a common risk factor for acute pancreatitis?

A. Hypertriglyceridemia
B. Gallstones
C. Hypercalcemia
D. Smoking
E. Alcohol

Answer: D

Chronic Pancreatitis

Definition

- characterized by long-standing inflammation & **fibrosis** then destruction of the exocrine Pancreas parenchyma, followed by loss of the endocrine parenchyma in the late stages
- The chief distinction from acute pancreatitis is the irreversible impairment in pancreatic function that is characteristic of chronic pancreatitis.

Etiology

There is significant overlap in the causes of acute and chronic pancreatitis

- Tropical pancreatitis: which is a poorly characterized disease seen in Africa & Asia, it has also been attributed to malnutrition.
- Long term Chronic alcohol abuse (**the most common cause**)
- Repeated acute pancreatitis (Long-standing obstruction of the pancreatic duct by pseudocysts, trauma , pancreas divisum, calculi or neoplasms)
- Idiopathic chronic pancreatitis (in 40% of cases)
- Hypercalcemia & hyperlipidemia
- Hereditary pancreatitis (Germline mutations in genes such as CFTR) (PRSS1 mutations)
- Autoimmune injury to the gland (IgG4-related disease)
- Biliary tract disease, are usually middle-aged males (2nd most common cause)

Epidemiology

Prevalence ranges between 0.04 - 5% of the U.S. population

Male Slides

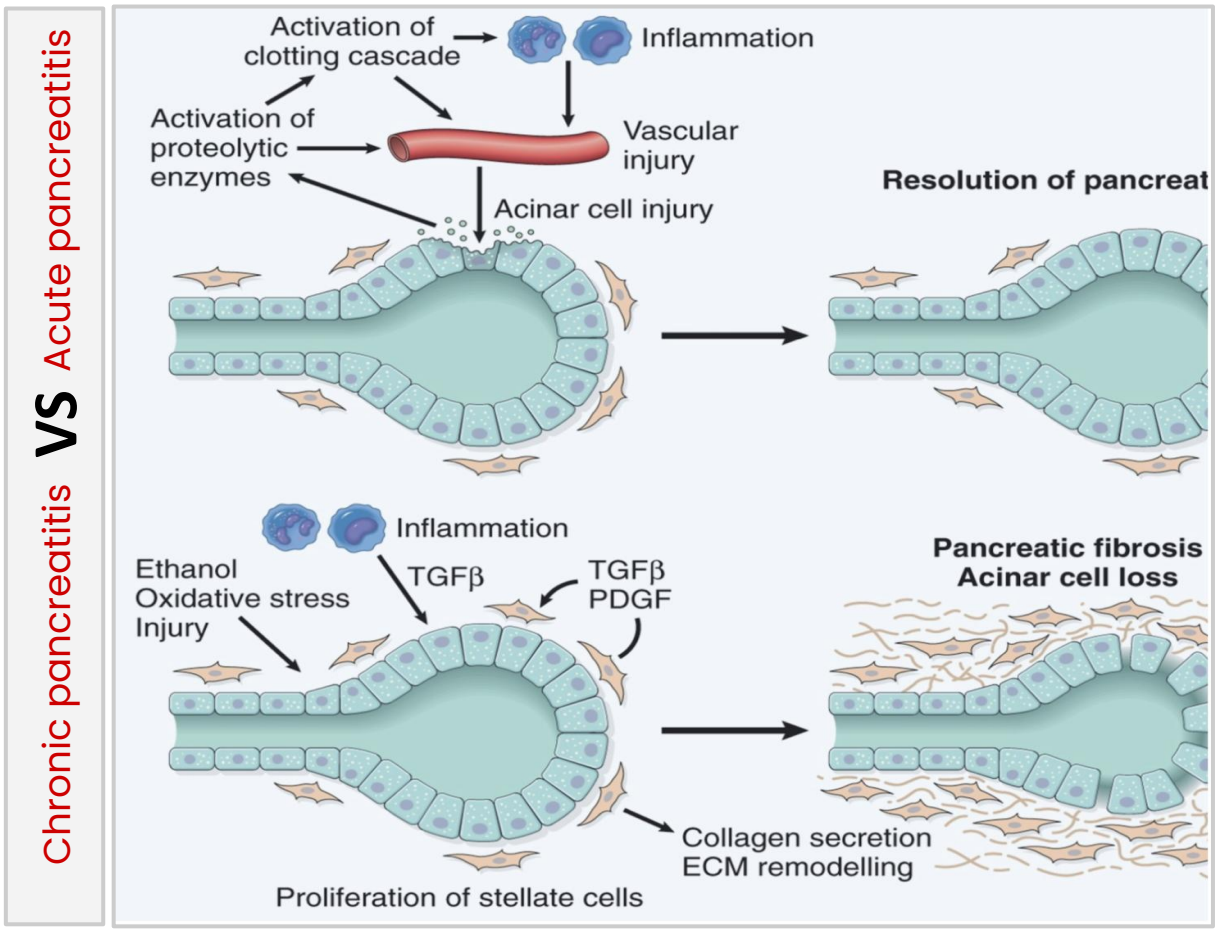
pancreatitis and:

Hypercalcemia	Hyperlipidemia
• Conversion of trypsinogen to trypsin. • The accumulation of calcium can promote the formation of ductal obstruction, pancreatic calculi.	• Might impair circulatory flow in capillary beds lead to ischemia. • Triglycerides and free fatty acids cause edema, hemorrhage and elevated amylase levels.

Chronic Pancreatitis

Pathogenesis

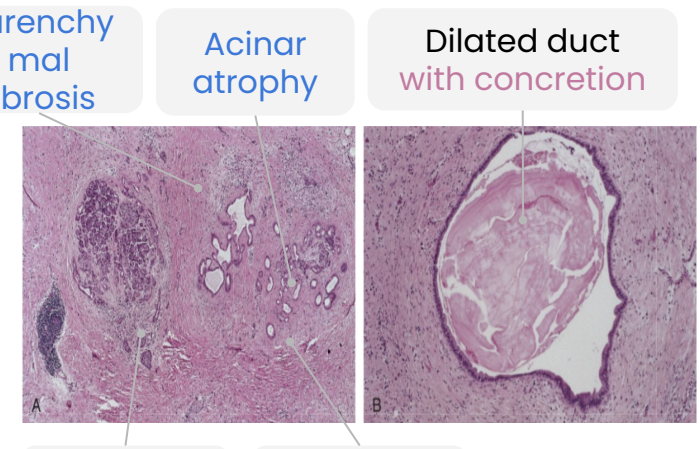
Arises from precursor lesions (PanINs) as a result of progressive accumulation of inherited and acquired genetic mutations in pancreatic epithelium.



Morphology

Female Slides

1. Parenchymal **fibrosis**.
2. Reduced number & size of acini (acinar loss).
3. **Variable dilation of the pancreatic ducts.**
4. Relative sparing of the islets of Langerhans (+/- hyperplasia).
5. Chronic inflammatory infiltrate around remaining lobules and ducts.
6. Ductal epithelium may be atrophied, hyperplastic or exhibit squamous metaplasia.
7. Ductal concretions may be seen.



Chronic Pancreatitis

Clinical Features

Female Slides

Present in several different ways:

- Repeated bouts of jaundice.
- Vague indigestion.
- Persistent or recurrent abdominal and back pain.
- Complication: pancreatic insufficiency (malabsorption led to wt. loss & hypoalbunemic edema) & DM develop.

Female Slides

Attacks can be precipitated by:

- Alcohol abuse.
- Overeating (increases demand on pancreatic secretions).
- Drugs as opiates (increases the muscle tone of the sphincter of Oddi).

Male Slides

Silent or repeated attacks of abdominal pain, or persistent abdominal and back pain. Attacks may be precipitated by alcohol abuse, overeating (which increases demand on the pancreas), or the use of opiates and other drugs.

Male Slides

During an attack of abdominal pain, there may be mild fever and mild-to-moderate elevations of serum amylase.
Calcifications can be seen within the pancreas by CT scan and ultrasonography.
• Complications: Severe pancreatic exocrine insufficiency, chronic malabsorption, diabetes mellitus (due to destruction of islets of Langerhans),and pancreatic pseudocysts.

Female Slides

Diagnosis requires a high degree of clinical suspicion.
• During attack of abdominal pain, there may be mild fever & moderate elevation of serum amylase.
• CT or ultrasonography: visualization of **calcifications** within the pancreas.

Prognosis

Female Slides

10% develop **pancreatic pseudocysts.**

- Long-term prognosis is poor: 50% mortality rate over 20 to 25 years.
- Persons with hereditary pancreatitis have a 40% lifetime risk of developing pancreatic can

Chronic pancreatitis

Diagnosis

Amylase in chronic pancreatitis:
- Less reliable than in acute disease.
- Values are variable: either normal, borderline, or slightly increased.

Lipase in chronic pancreatitis: ✔ Not clinically useful.

Serum immunoreactive trypsin in chronic pancreatitis: Decreased concentration.

Summary

Acute pancreatitis	Chronic pancreatitis
• Gallstone alcohol. • **Activation of trypsinogen.** • Fat necrosis, hemorrhage. • Acute abdomen, hypotension/shock, hypocalcaemia. • Serum amylase.	• long-term alcohol abuse and biliary tract disease. • Recurrent abdominal pain. • Fibrosis, Acinar atrophy. • Serum amylase mild. • Irreversible impairment in pancreatic function, chronic malabsorption, diabetes mellitus .

Deep Focus Question

With which gammopathy is chronic pancreatitis associated?

A. IgM
B. IgE
C. IgG
D. IgA
E. IgD

Answer: C

Deep Focus Question

What is a dietary recommendation for patients with chronic pancreatitis?

A. Small, infrequent, low-fat meals
B. Large, infrequent, low-fat meals
C. Small, frequent, low-fat meals
D. Small, frequent, low-carbohydrate meals

Answer: C

Keywords

Acute Pancreatitis	• Reversible • Biliary tract diseases & Gallstones • Alcoholism (more common)& Hypercalcemia (as a cause) • Mutations in the cationic trypsinogen (PRSSI) , and • trypsin inhibitor (SPINKI) genes • local fat necrosis. • Lysosomal hydrolyse • edema & Acute inflammation (neutrophils) • interstitial hemorrhage (activation of elastases) • Grossly : Red-black hemorrhage interspersed with foci of yellow-white, chalky fat necrosis • Epigastric pain & steatorrhea (in both) • Shock & renal failure • acute respiratory distress syndrome • elevated serum amylase • elevated serum lipase (more specific) • Hypocalcemia (as symptom not cause) • Activation of trypsinogen into trypsin (main pathology for pancreatitis in general)
	Pseudocyst of pancreas : • localized collections of liquefied areas of necrotic-hemorrhagic fluid material rich in pancreatic enzymes • cysts lack an epithelial lining • Seen in alcoholic pancreatitis & trauma • predispose to intraperitoneal hemorrhage or peritonitis. • Could be seen in chronic
Chronic Pancreatitis	• long-standing inflammation & irreversible impairment • Parenchymal fibrosis • Repeated bouts of acute pancreatitis • Chronic alcohol abuse • Hereditary pancreatitis (Germline mutations in genes CFTR which includes the PRSS1 mutations) • Arises from precursor lesions (PanINs) • Relative sparing of the islets of Langerhans • acinar loss • squamous metaplasia in Ductal epithelium • dilation of the pancreatic ducts • May be due to jaundice • opiates • moderate elevation of serum amylase or within normal • exocrine insufficiency • calcifications • Normal lipase • Serum immunoreactive trypsin in chronic pancreatitis has decreased concentrations.

IF YOU WANT A SUMMARY CLICK HERE

YOU Vs MCQs

1

Which mechanism plays a role in human acute pancreatitis caused by ischemia, viral infections, drugs, and trauma?

A- Pancreatic duct obstruction

B- Primary acinar cell injury

C- Defective intracellular transport of proenzymes

D- Activation of elastases

2

What are the basic alterations seen in the histopathology of acute pancreatitis?

A- Inflammation and edema

B- Parenchymal necrosis

C- Fat necrosis

D- All the above

3

What is the primary triggering event in acute pancreatitis?

A- Hyperactivation of trypsinogen

B- Alcohol consumption

C- Gallstone impaction

D- Ischemia

4

What is the most common cause of chronic pancreatitis?

A- Gallstones

B- Alcoholism

C- Trauma

D- Autoimmune disorder

1-B / 2-D / 3-A / 4- B

YOU Vs MCQs

5

What will be the laboratory findings in Acute pancreatitis?

A- ↓ Serum amylase

B- Hypercalcemia

C- ↓ Serum lipase[2] levels

D- Hypocalcemia

6

Which of the following is NOT a morphology of Chronic pancreatitis ?

A- Variable dilation of the pancreatic ducts

B- Parenchymal fibrosis

C- ↑ Acinar cells

D- ↓Islets of Langerhans

7

Which genetic mutations are associated with recurrent severe acute pancreatitis?

A- Mutations in the cationic trypsinogen and trypsin inhibitor genes

B- Mutations in the insulin and glucagon genes

C- Mutations in the lipase and amylase genes

D- Mutations in the somatostatin and gastrin genes

5-D / 6-C / 7- A

YOU VS CASES

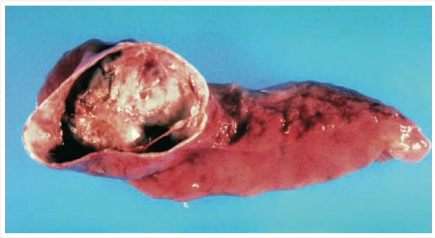
1. A 42-year-old obese woman (BMI = 32 kg/m2) presents with severe abdominal pain that radiates to the back. There is no history of alcohol or drug abuse. The blood pressure is 90/45 mm Hg, respirations are 32 per minute, and pulse is 100 per minute. Physical examination shows abdominal tenderness, guarding, and rigidity. An X-ray film of the chest shows a left pleural effusion. Laboratory studies reveal elevated serum amylase (850 U/L) and lipase (675 U/L), and hypocalcemia (7.8 mg/dL). Which of the following is the most likely diagnosis?			
A.Acute cholecystitis	B.Acute pancreatitis	C.Alcoholic hepatitis	D.Chronic calcifying pancreatitis
2.Which of the following is most likely associated with the pathogenesis of the condition of the patient described in Question 1?			
A.Carcinoid syndrome	B.Cholelithiasis	C.Insulinoma	D.Pancreatic adenocarcinoma
3.A 60-year-old alcoholic man presents with a 6-month history of recurrent epigastric pain, progressive weight loss, and foul smelling diarrhea. The abdominal pain is now almost constant and intractable. An X-ray film of the abdomen reveals multiple areas of calcification in the mid-abdomen. Which of the following is the most likely diagnosis?			
A.Carcinoid syndrome	B.Chronic pancreatitis	C.Crohn disease	D.Insulinoma
4.Which of the following findings is most likely to be encoun-tered in the patient described in Question 3?			
A.Achlorhydria	B.Hypoglycemia	C.Melena	D.Steatorrhea
5. A 50-year-old woman complains of persistent abdominal pain, anorexia, and abdominal distention. Her past medical history is significant for a previous hospitalization for acute pancreatitis. Physical examination shows jaundice and a non pulsatile abdominal mass. Laboratory studies reveal normal serum levels of bilirubin, AST, and ALT. A CT scan of the abdomen shows a fluid-filled cavity in the head of the pancreas. What is the most likely diagnosis?			
A.Acute hemorrhagic pancreatitis	B.Insulinoma	C.Pancreatic islet cell tumor	D.Pancreatic pseudocyst

1-B / 2-B / 3-B/ 4-D / 5-D

NEED EXPLANATION ? CLICK HERE

YOU Vs CASES

6.The surgical specimen is shown in the image for the patient described in Question 5. In addition to blood and necrotic debris, which of the following best describes the contents of this cystic lesion?



A.Bile	B.Chylous fluid	C.Lymph	D.Pancreatic enzymes

7.A 38-year-old woman presents to the emergency department with nausea and progressive right upper quadrant abdominal pain for the past day. For the past year, she has had occasional pain in her right upper quadrant that often goes away on its own after a few hours. She was recently diagnosed with multiple gallstones, for which she underwent an elective uncomplicated endoscopic retrograde cholangiopancreatography (ERCP) 3 days ago. Her past medical history is otherwise unremarkable. On physical examination, there is tenderness over the epigastrium with no guarding or rebound. Her blood pressure is 110/68 mm Hg, pulse 98/min, temperature 36.2°C (97.2°F), and respiratory rate 11/min. Laboratory test results are pending. An imaging study of the abdomen confirms the most likely diagnosis. Which of the following is most likely to be below the normal range in her blood due to her current condition?

A.Trypsinogen	B. C-reactive protein	C.Calcium	D.lipase

8.A 29-year-old woman presents with a 2-hour history of sudden onset of severe mid-epigastric pain. The pain radiates to the back and is not relieved by over-the-counter antacids. The patient also complains of profuse vomiting. The patient's medical history is negative for similar symptoms. She consumes 3–4 alcoholic drinks daily. The blood pressure is 80/40 mm Hg and the heart rate is 105/min. Examination of the lungs reveals bibasilar crackles. Abdominal examination reveals diffuse tenderness involving the entire abdomen, marked guarding, rigidity, and reduced bowel sounds. The chest X-ray is normal. However, the abdominal CT scan reveals peritoneal fluid collection and diffuse pancreatic enlargement. The laboratory findings include:

Aspartate aminotransferase	63 IU/L
Alkaline phosphatase	204 IU/L
Alanine aminotransferase	32 IU/L
Serum amylase	500 IU/L (Normal: 25-125 IU/L)
Serum lipase	1,140 IU/L (Normal: 0-160 IU/L)
Serum calcium	8.0 mg/dL

A.Coagulative necrosis	B.Fat necrosis	C.Caseous necrosis	D.Dry gangrene

6-D / 7-C / 8-B

NEED EXPLANATION ? CLICK HERE

YOU Vs CASES

EXTRA CASES MAY REQUIRE EXTRA INFO

1.A 44-year-old man comes to the emergency department with abdominal pain and nausea for the past 24 hours. The pain started a few hours after dinner, and he describes it as a constant pain in the upper part of his abdomen that radiates to his back and flanks. The patient also reports nausea, intermittent vomiting, and abdominal distention. Family history is significant for recurrent episodes of pancreatitis in his father and paternal uncle. He reports drinking an average of 2 alcoholic drinks per week. His temperature is 37.7°C (99.9°F), pulse is 92/min, respirations are 20/min, peripheral oxygen saturation is 96%, and blood pressure is 149/94 mmHg. He appears distressed and anxious. Abdominal examination shows tenderness and rigidity over the epigastric and periumbilical regions. Bowel sounds are diminished. No bruising of the umbilicus or flanks are seen. Which of the following laboratory results is the most specific in diagnosing this patient's condition?

A.Blood urea nitrogen (BUN) level > 22 mg/dL	B.Elevated blood alcohol level	C.Serum amylase > 3 times the upper limit of normal	D.Serum lipase > 3 times the upper limit of normal

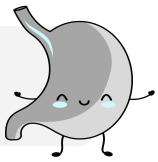
2.A 58-year-old woman with a history of chronic alcohol use disorder comes to the office because of diarrhea, generalized weakness, and a 6.8-kg (15-lb) weight loss over the past 6 months. She reports intermittent dull upper abdominal pain that will last for days at a time and is not improved with antacids. After meals, she feels that her abdomen is distended. She characterizes her multiple daily bowel movements as greasy, foul-smelling, and oily. She recently was the driver involved in a minor traffic accident, which she attributes to worsening eyesight at night. Her temperature is 37.0°C (98.6°F), pulse is 78/min, respirations are 16/min, and blood pressure is 135/85 mmHg. Abdominal examination shows resonance to percussion throughout and a mildly tender epigastrium. Bowel sounds are hyperactive. Laboratory values show the following:

Laboratory value	Result
Calcium, serum	7.6 mg/dL
Partial thromboplastin time	60 seconds
Prothrombin time	28 seconds

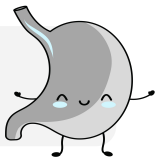
A.Chronic gastritis	B.Celiac disease	C.Pancreatic adenocarcinoma	D.Chronic pancreatitis

1-D / 2-D

Pathology Team



Leader زياد العتيبي



Leader لمى العتيبي

- عائشة إبراهيم
- الدانة عبدالله
- ريما المطيري
- زياد حكمي
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- لؤي الحديثي
- محمد السلامه
- يوسف بادغيش
- أفنان الأحمري
- هدى بن جدعان
- محمد العرفج
- نوره المالك
- شوق الخليفة
- عبدالرحمن المسلّم
- رغد المصلح
- فيصل الشويعر
- سلطان البقمي
- عبدالله الضويحي
- هيا الزير
- معاذ الحضيف
- ساره العجاجي
- رند ابا الخيل
- منصور العتيبي
- دانه المحيسن
- دينا المهوس
- نورة المحيميد
- ليان الرويلي
- ريماز المحمود
- عروب المحمود
- الجوهرة الوهيبي
- عبدالله الكودري
- إيلاف معتبي
- يزيد ال طلحه
- رزان السطيحي
- زياد السويلم
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