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03

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GNT pathology cases file
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Pathology

Acute and Chronic Pancreatitis

439

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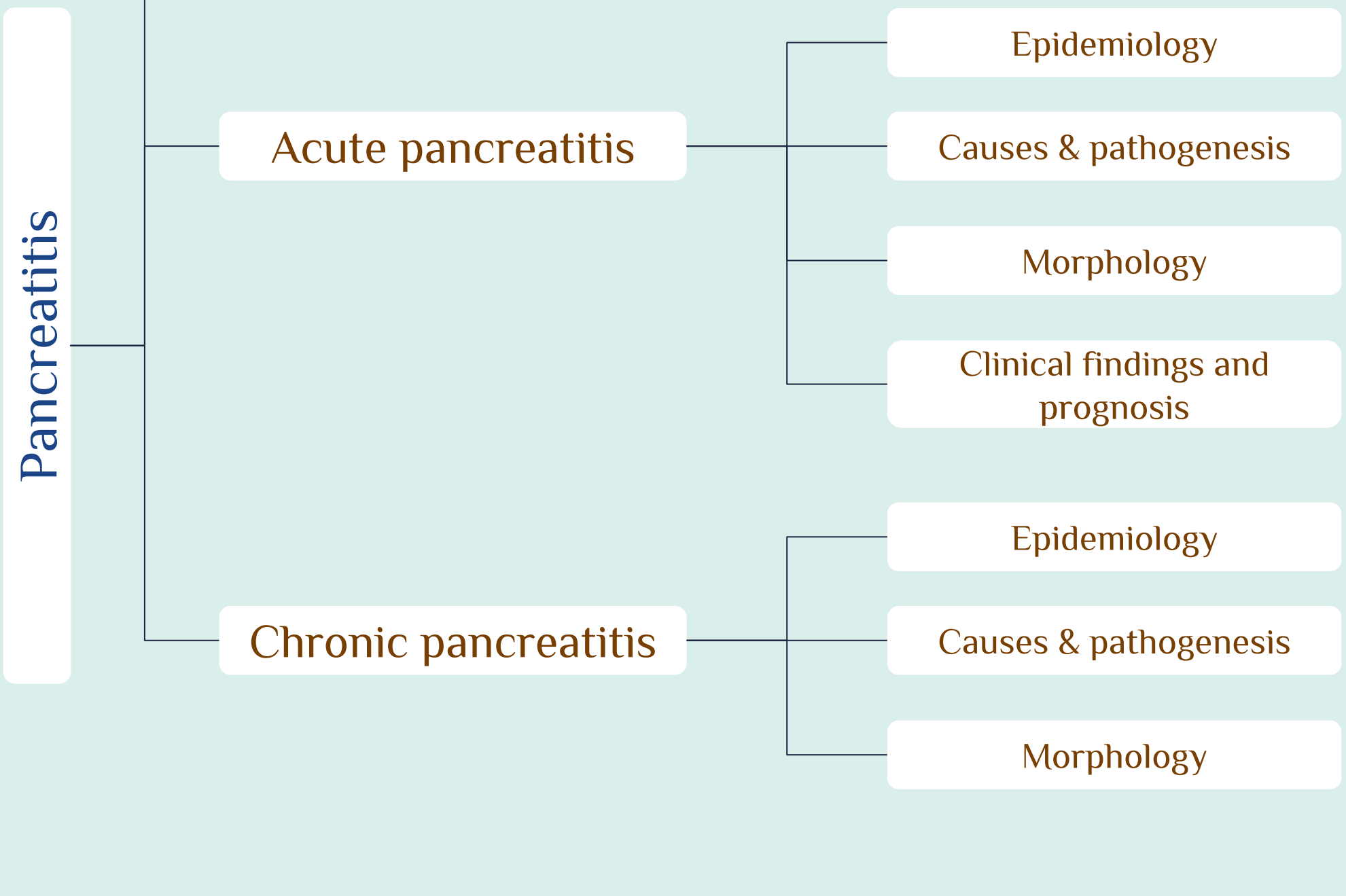
- Important
- Doctor's note
- Extra info
- Main text
- ★ Male's slide
- ★ Female's slide

"اللهم لا سهل الا ماجعلته سهلا وانت تجعل الحزن إذا شئت سهلا"

Objective

01 Describe the definition, epidemiology, pathogenesis, morphology, clinical findings , and possible complication of acute and chronic pancreatitis

Overview



Pancreatitis

Helpful video

Overview

The pancreas is really two organs packaged into one.

❖ **Endocrine portion (10% of pancreas)**

-Islets of Langerhans : secrete **insulin**, **glucagon**, and somatostatin.

-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** and the ductules and ducts that convey their secretions to the duodenum.

-Pancreatitis : encompasses a group of disorders characterized by **inflammation of the pancreas.**

-The clinical manifestations can range in severity from a mild, self-limited disease to a life-threatening acute inflammatory process.

❖ **Acute pancreatitis** the gland can return to normal if the underlying cause of the pancreatitis is removed , **reversible** .

❖ **Chronic pancreatitis** is defined by the **irreversible** loss of exocrine pancreatic parenchyma.

Acute pancreatitis

Helpful video

Definition[1]

❖ **Reversible** pancreatic parenchymal injury associated with **inflammation**.

Epidemiology

❖ Acute pancreatitis is relatively common, with an annual incidence rate in **Western countries** of 10 to 20 cases per 100,000 people .

❖ Biliary tract diseases (stones) 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[2] is :

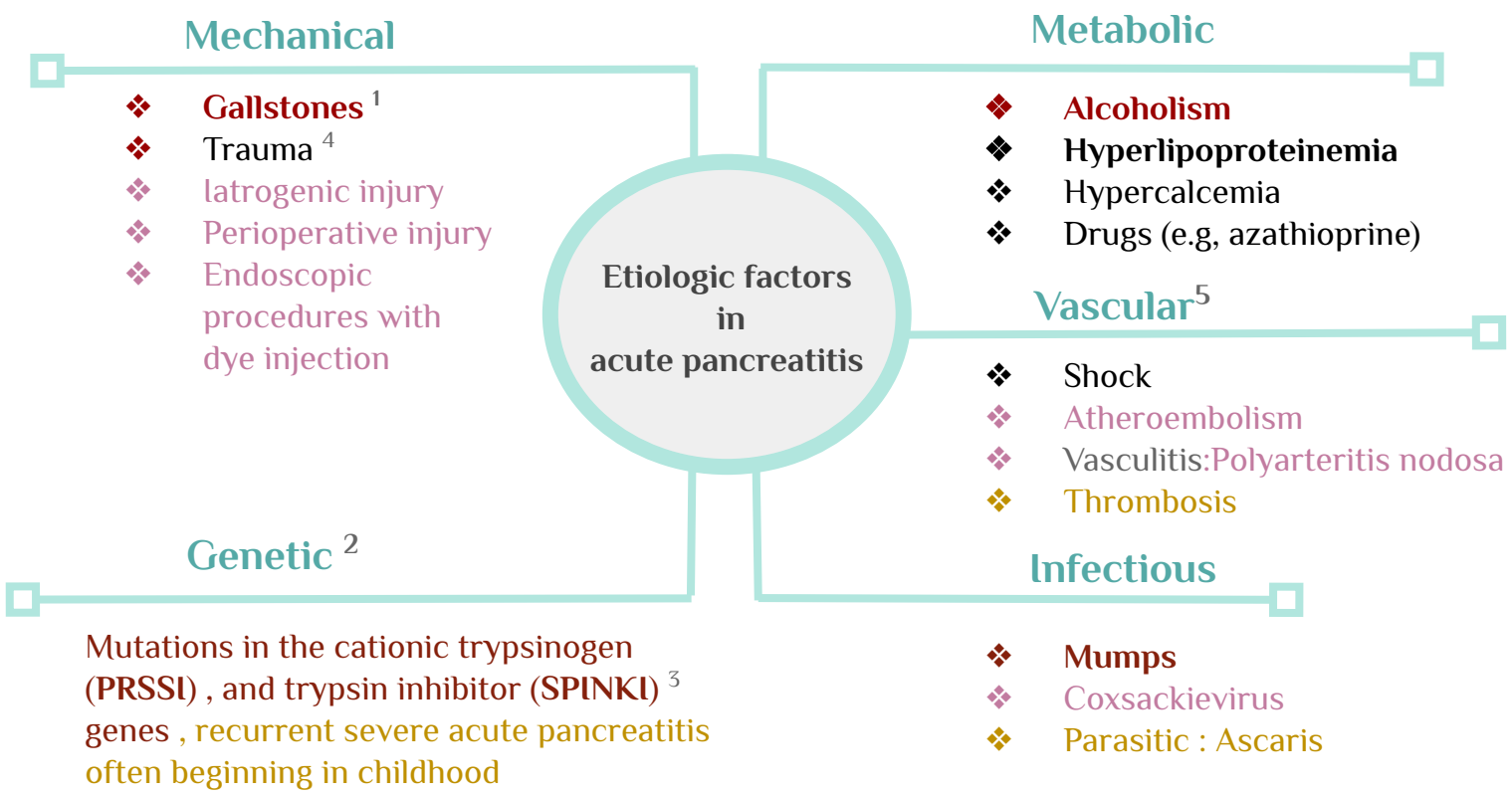
➢ 1 : 3 in the group with biliary tract diseases

➢ 6 : 1 in those with alcoholism .

1-Is a necro-inflammatory disease that is characterized by infiltration of the pancreas by inflammatory cells and destruction of the pancreatic exocrine cells

2-Alcoholism more in males, Stones more in females

Etiology



Pathogenesis

- Acute pancreatitis appears to be caused by autodigestion of the pancreas by **inappropriately activated pancreatic enzymes**. The pancreas is normally protected from autodigestion by synthesis of pancreatic enzymes in the acinar cells in the **proenzymes** form

1.Pancreatic duct obstruction	❖ blocks ductal flow ➔ ↑ 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 flow ➔ vascular insufficiency & ischemic injury to acinar cells
2. Primary acinar cell injury	❖ This pathogenic pathway comes into play in acute pancreatitis caused by ischemia, viral infections, drugs & direct trauma to the pancreas
3. Defective Intracellular transport of proenzymes within acinar cells	❖ 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

1-Mechanism of gallstones: impact of the stones within the duct leading to obstruction of duct depending on the floor of pancreatic enzyme for the ampulla of vater , obstruction leads to accumulation of pancreatic enzymes
2-hereditary pancreatitis
3-premature activation of trypsinogen into trypsin
4-sharp trauma mainly most in children « seat belt »
5-decreased blood supply

The histologic changes seen in acute pancreatitis strongly suggest autodigestion of the pancreatic substance by inappropriately activated pancreatic enzymes. As described previously, the zymogen forms of pancreatic enzymes must be enzymatically cleaved to be activated; trypsin is central in this process, so **activation of trypsin** / trypsinogen is a critical triggering event in acute pancreatitis. If trypsin is inappropriately generated from its proenzyme trypsinogen, it can activate itself as well as other proenzymes (e.g. phospholipases and elastases) that can then take part in the process of autodigestion. Trypsin also converts prekallikrein to its activated form, thus sparking the kinin system, and, by activation of factor XII (Hageman factor), also sets in motion the clotting and complement systems

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

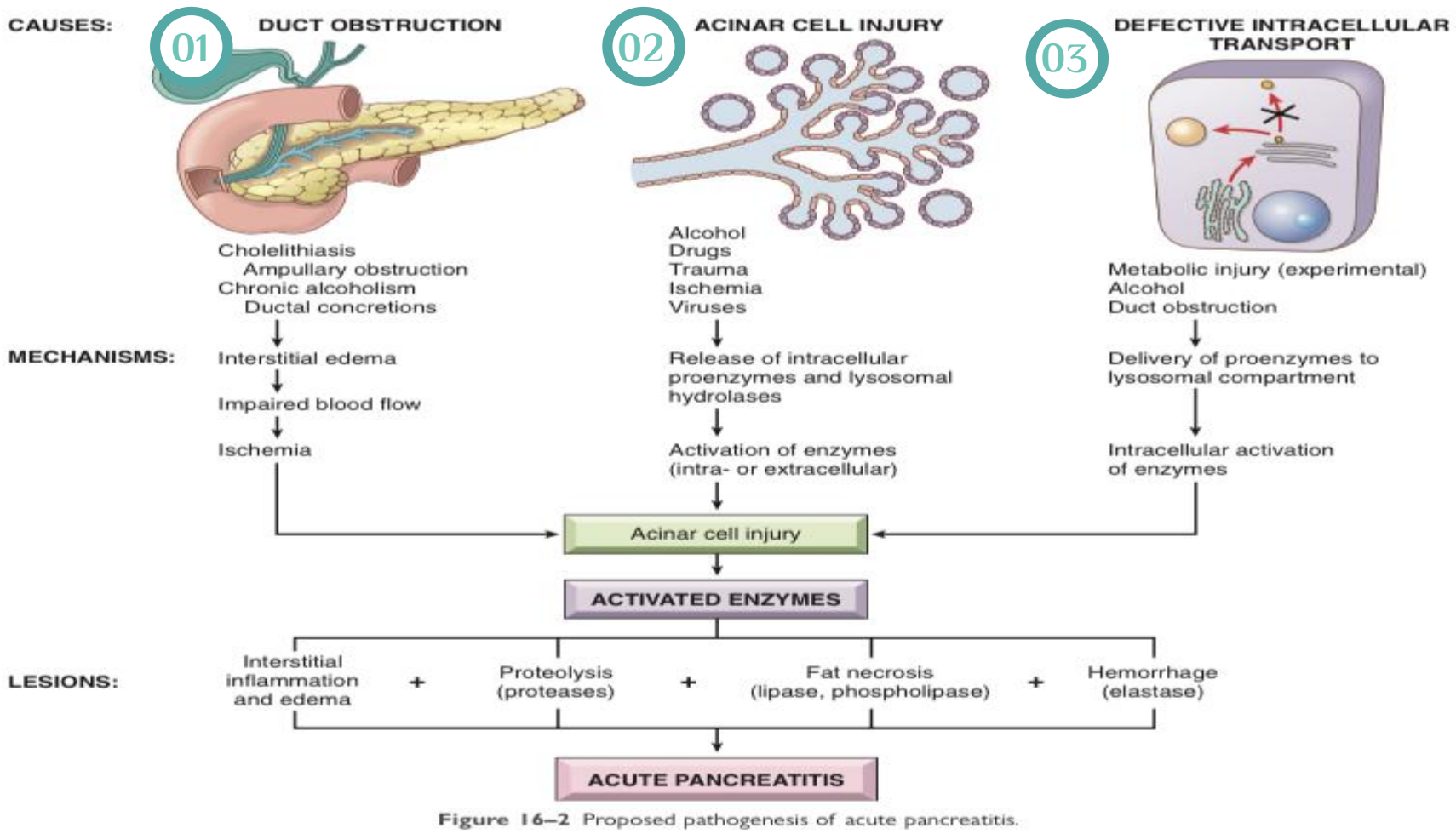


Figure 16–2 Proposed pathogenesis of acute pancreatitis.

01 **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.

02 **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.

03 **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. However, at least

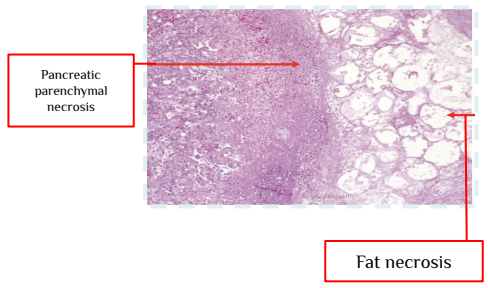
Alcohol consumption may causes pancreatitis by several mechanisms :

- Alcohol transiently increases pancreatic exocrine secretion & contraction of the sphincter of Oddi
- Alcohol has direct toxic effects on acinar cells
- Chronic alcohol ingestion ➔ secretion of protein-rich pancreatic fluid ➔ deposition of inspissated protein plugs➔ obstruction of small pancreatic ducts

Morphology

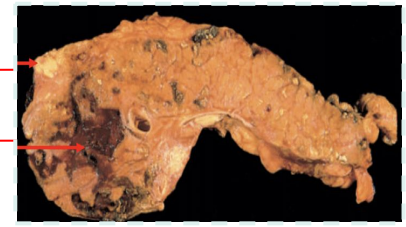
❖ The morphology of acute pancreatitis ranges from inflammation and edema to severe extensive necrosis & hemorrhage

Histopathologic Morphology

★ The basic alterations are	Acute necrotizing pancreatitis (sever form)	Hemorrhagic pancreatitis (most severe form)
1. Microvascular leakage causing **edema** **2.** **Necrosis of fat** by lipolytic enzymes (lipases) 3. **Acute inflammation** 4. **Proteolytic destruction** of **pancreatic parenchyma** by proteases 5. Destruction of blood vessels and subsequent **interstitial hemorrhage** due to activation of elastases	❖ 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 & bowel mesentery, and even outside the abdominal cavity (e.g, in subcutaneous fat) ❖ Peritoneum usually contains a serous, slightly turbid, brown-tinged fluid with globules of fat (derived from enzymatically digested adipose tissue)	❖ Extensive parenchymal **necrosis** and diffuse **hemorrhage** leading to shock 

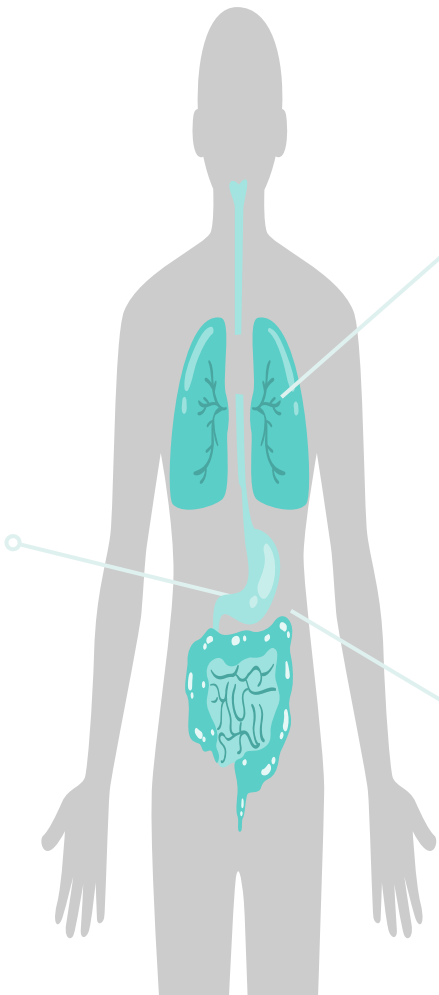
Gross Morphology

❖ Red-black hemorrhage interspersed with foci of yellow-white, chalky fat necrosis
❖ Foci of necrosis may also be found in extra-pancreatic collections of fat
❖ Fat necrosis results from enzymatic destruction of fat cells, the released fatty acids combine with calcium to form insoluble salts that precipitate in situ .



Clinical Features

❖ **Abdominal pain** : varies in severity from mild & uncomfortable to sever & incapacitating
❖ Characteristically, the pain is constant, intense & often is referred to **upper back**
❖ Full-blown acute pancreatitis constitutes a **medical emergency** "**acute abdomen**": sudden onset of abdominal pain, abdominal guarding/rigidity & absence of bowel sounds
❖ It must be differentiated from : Ruptured acute appendicitis, perforated peptic ulcer, acute cholecystitis with rupture & occlusion of mesenteric vessels with infarction of bowel
❖ 80% of cases are mild & self limiting; remaining 20% develop severe disease ➔ shock, hemorrhage & chronic pancreatitis



Consequence of acute pancreatitis later on :

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

↑Microvascular permeability → hypovolemia + endotoxemia (from breakdown of the barriers between gastrointestinal flora and bloodstream) → peripheral vascular collapse (**shock**) → acute tubular necrosis in kidney → acute **renal failure**

Laboratory findings

Laboratory findings include markedly **elevated serum amylase**[1] in 1st 24 hr, followed (within 72-96 hr) by rising **serum lipase**[2] **levels**

Hypocalcemia[3] (due to precipitation of Ca^{+2} in areas of fat necrosis) If persistent, it is a poor prognostic sign

CT scan or MRI will show enlarged & inflamed pancreas & inflammation of peripancreatic tissue

Management :
Supportive therapy: such as maintaining BP & alleviating pain
"Resting" the pancreas by total restriction of food & fluids
Treat the primary cause

Prognosis

- Most patients will eventually recover
- 5% can die from shock during the first week of illness
- Some may develop acute respiratory distress syndrome +/- acute renal failure (fatal complications)
- Survivors may develop (complication): 1- Sterile or infected pancreatic "abscesses" 2- Pancreatic pseudocysts

only found in the boy's slides

Mild self-limiting ⟷ **Life threatening process**

Return to normal if underlying cause is removed

Common Causes	**80%**	**Biliary tract disease**	**Gallstones**
		Alcoholism	

Less Common Causes	Obstruction of the pancreatic duct system	
	Medications	Sulfonamides , azathioprine
	Metabolic disorders	Hypercalcemia, hyperlipoproteinemia
	Acute ischemia	
	Trauma	
	Genetic	**PRSS1 SPINK1** : recurrent severe acute pancreatitis often beginning in childhood

1-Non-specific as it is secreted from the pancreas and salivary glands
2-more specific because it's only secreted from the pancreas
3-the worse the inflammation, the lower serum calcium level which predict a worse prognosis

Chronic pancreatitis

Helpful video

Definition

- Chronic pancreatitis is characterized by long-standing inflammation & fibrosis then destruction of the exocrine parenchyma, followed by loss of the endocrine parenchyma in the later stages[1].
- The chief distinction from acute pancreatitis is the irreversible impairment in pancreatic function that is characteristic of chronic pancreatitis.
- The fibrosis, destruction, and atrophy of parenchyma is irreversible in chronic pancreatitis and these features also differentiates it from acute pancreatitis.

Epidemiology

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

Etiology

01 Repeated bouts of acute pancreatitis (Long-standing obstruction of the pancreatic duct by pseudocysts, trauma , pancreas divisum, calculi or neoplasms)

02 **Chronic alcohol abuse (the most common cause)**

03 Biliary tract disease such as gallstones, and these patients are usually middle-aged
males. (Second most common cause)

1-in the late stages, associated with pancreatic insufficiency, steatorrhea, diabetes due to destruction of islet- beta cells, pancreatic calcification, and fibrosis.

Etiology

Less common causes include:

04 Hypercalcemia & lipidemia

05 Tropical pancreatitis: a poorly understood disorder seen in Africa & Asia, with a subset of cases having genetic basis, it has also been attributed to malnutrition

06 Hereditary pancreatitis (Germline mutations in genes such as CFTR which includes the **PRSS1 mutations)**

07 Autoimmune injury to the gland (lgG-related disease)

08 Idiopathic chronic pancreatitis (in 40% of cases)

Pathogenesis

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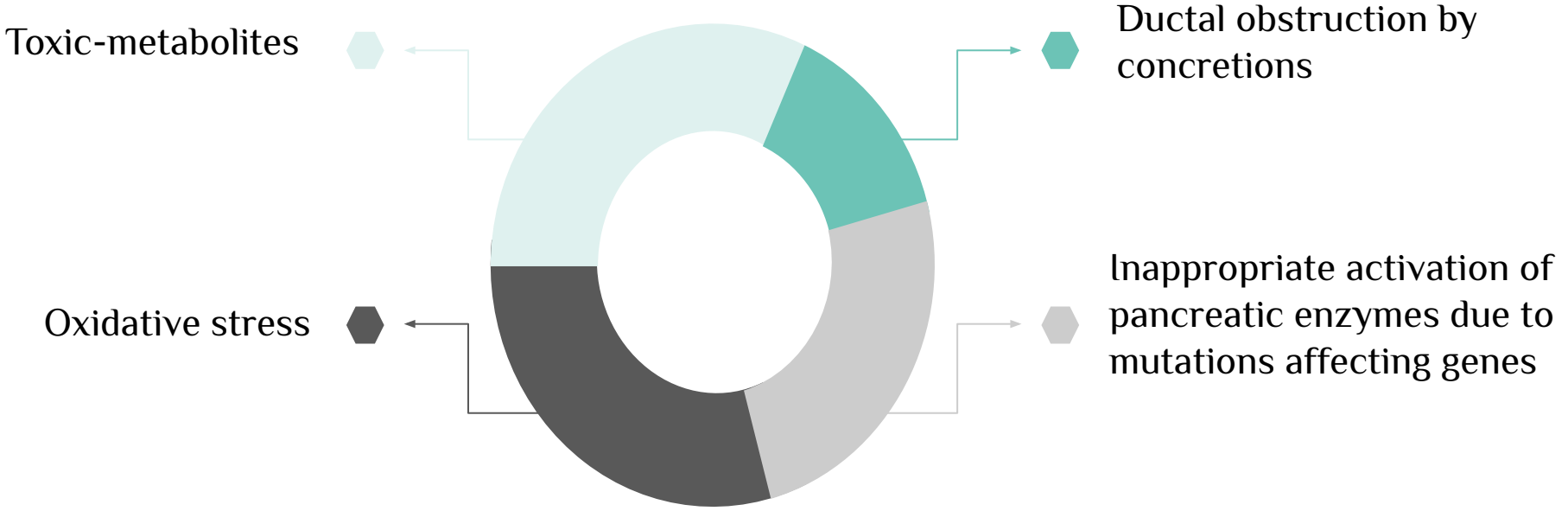
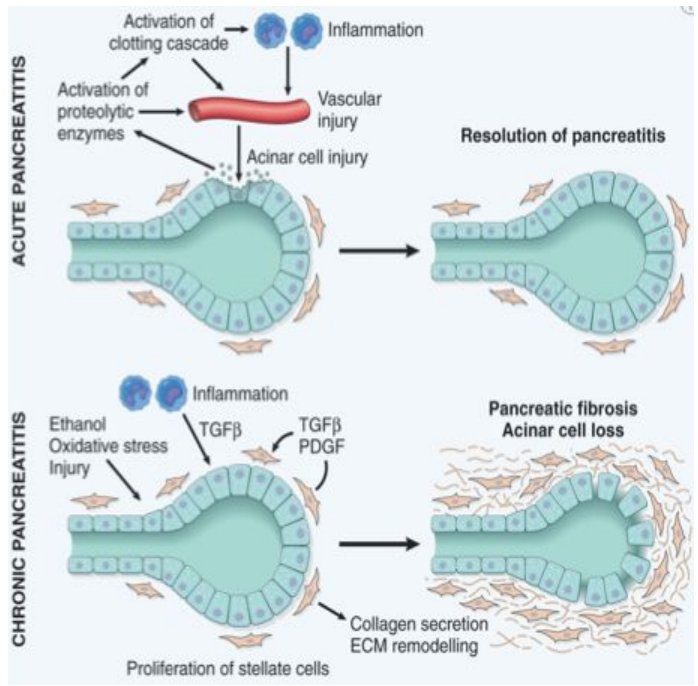
- Although the pathogenesis of chronic pancreatitis is not well defined, several hypotheses are proposed:



- Repeated episodes of acinar cell injury lead to the production of TGF-β and PDGF, resulting in proliferation of myofibroblasts, secretion of **collagen** and irreversible **loss of acinar cell** mass, **fibrosis**, and **pancreatic insufficiency**

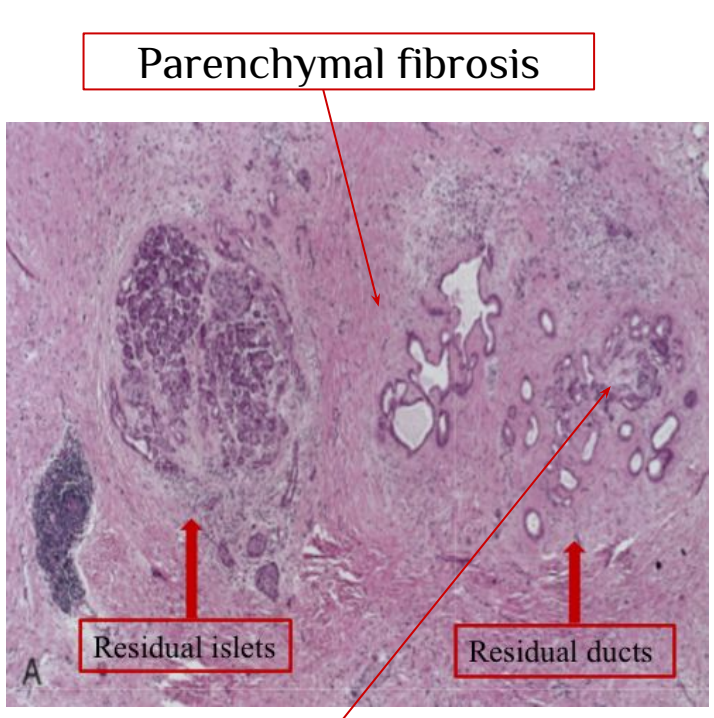
- acute: Distraction of the acinar epithelium=>secondary inflammation =>affection of blood vessels =>hemorrhage and edema but the parenchyma itself does not affected => the affected acinar can be regenerated and back to normal
chronic: ethanol/oxidative stress/injury to the epithelium=>inflammation => release TGF beta and PDGF => proliferation of stellate cells around the acinar cell=> collagen secretion and ECM remodulation=>calcification and fibrosis =>pancreatic insufficiency

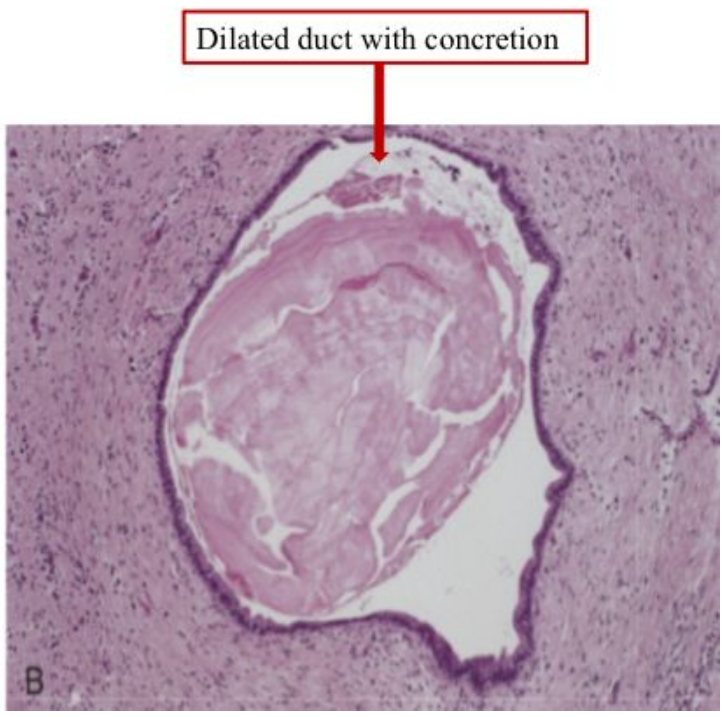


Morphology

Chronic pancreatitis is characterized by:

1. Parenchymal fibrosis
2. Reduced number & size of acini (acinar loss) and will also have less secretions
3. Variable dilation of the pancreatic ducts which occurs due to the fibrosis and destruction causing obstruction and accumulation
4. Relative sparing of the islets of Langerhans, eventually will disappear as well
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





relative sparing of the islets of Langerhans,

The endocrine portion of the pancreas withstands more than the exocrine pancreas (Endocrine portion is less susceptible to changes than exocrine portion)

Clinical Features

Chronic pancreatitis presents in several different ways:

1) Repeated bouts of jaundice
2) Vague indigestion
3) Persistent or recurrent abdominal and back pain

- Or it may be entirely silent until pancreatic insufficiency (malabsorption leading to weight loss & hypo-albunemic edema) & DM[1] develop

- Attacks can be precipitated by:

1) Alcohol abuse
2) Overeating (increases demand on pancreatic secretions)
3) Drugs as opiates (increases the muscle tone of the sphincter of Oddi)

MALE DR: Sometimes patients present with back pain only without abdominal pain.

Another important feature is the foul smelling diarrhea caused by steatorrhea which occurs due to the malabsorption caused by the maldigestion which the pancreatitis leads to.

Pancreatitis leads to maldigestion which causes malabsorption.

Clinical picture and prognosis

- 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
- ~ 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 cancer
- Complications: Severe pancreatic exocrine insufficiency, chronic **malabsorption** (due to maldigestion), **diabetes mellitus** (due to destruction of islets of Langerhans), and pancreatic pseudocysts (which is also a complication of acute pancreatitis)

1-Type 1 diabetes mellitus (70% of cases)

Diagnosis

- Amylase in chronic pancreatitis is less reliable than in acute disease and the values are variable: either normal, borderline, or slightly increased
- Lipase in chronic pancreatitis is not clinically useful
- Serum immunoreactive trypsin in chronic pancreatitis has decreased concentrations.

Pseudocysts of pancreas

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- Pseudocysts are localized collections of necrotic-hemorrhagic fluid material rich in pancreatic enzymes. Such cysts lack an epithelial lining (hence the prefix "pseudo"), and they account for majority of cysts in the pancreas.
- Pseudocysts usually arise after an episode of acute pancreatitis, or of chronic alcoholic pancreatitis.
- Traumatic injury to the abdomen can also give rise to pseudocysts

Morphology pseudocysts of pancreas

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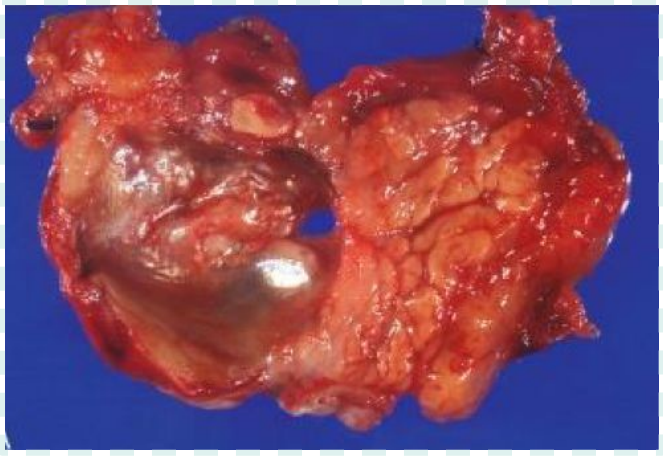
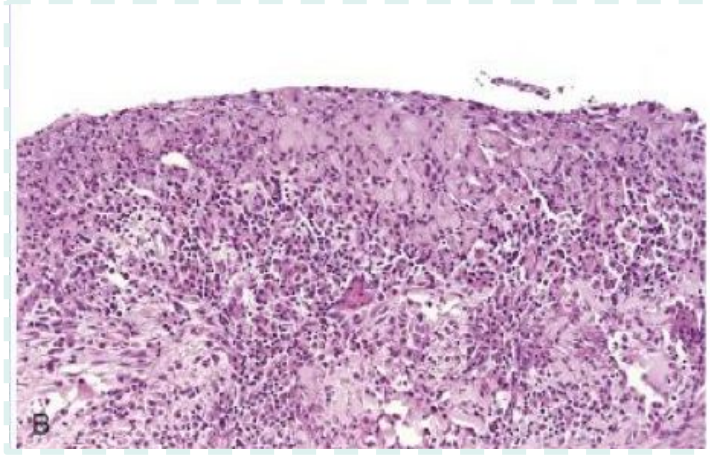
- Pseudocysts are usually **solitary**.

Pseudocysts can range in size from 2 (small) to 30 (very big) cm in diameter.

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

Large pseudocysts may also compress the peritoneal cavity.





Here we see inflammation, granulation tissue, but no epithelial lining and therefore it is a pseudocyst

Summary

	Acute pancreatitis	Chronic pancreatitis
Definition	Reversible pancreatic parenchymal injury associated with inflammation.	lrreversible injury of the pancreas leading to fibrosis, loss of pancreatic parenchyma, loss of exocrine and endocrine function, and high risk of developing pseudocysts.
Etiology	Caused by activation of pancreatic enzymes resulting in organ autodigestion; associated with alcohol, gallstones, hyperlipidemia, hypercalcemia, drugs (thiazides, sulfonamides), mumps infection, and autoimmune disease , Genetic factors (e.g., PRSS1, SPINK1).	ldiopathic or Caused by repeated bouts of pancreatic inflammation; associated with alcoholism and cystic fibrosis
Morphology	Gross: hemorrhagic areas with areas of white **fat necrosis.** Microscopic: interstitial **edema** and **inflammation**; necrosis of parenchyma with vascular damage .	Gross: **fibrotic** bands producing a lobular appearance; calcified concretions; pseudocyst formation. Microscopic: **destruction of acini** (islets of Langerhans spared) with fibrous replacement; mononuclear inflammatory infiltrate. -fibrosis , and acinar atrophy
Clinical feature	acute **abdominal pain radiating to the back**, systemic inflammatory response syndrome, and elevated serum lipase and amylase levels	intermittent or persistent abdominal pain , development of pancreatic insufficiency and **diabetes** , intestinal malabsorption.
Pathogenesis	The destructive changes in the pancreas are attributed to the liberation and activation of pancreatic enzymes. Though more than 20 enzymes are secreted by exocrine pancreas, 3 main groups of enzymes which bring about destructive effects on the pancreas are: 1. Proteases 2. Lipases and phospholipases 3. Elastases. The activation and release of these enzymes is brought about by one of the following mechanisms: i) Acinic cell damage ii) Duct obstruction iii) Block in exocytosis.	Repeated episodes of acinar cell injury leads to the production of TGF-β and PDGF, resulting in proliferation of myofibroblasts, secretion of collagen and irreversible loss of acinar cell mass, fibrosis, and pancreatic insufficiency.

QUIZ!

MCQs

01 | 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
02 | Which of the following is NOT seen in acute pancreatitis?			
A)fat necrosis	B)fibrosis	C)Eosinophils	D)Edema
03 | Which of the following is the most common cause of chronic pancreatitis.			
A) Recurrency of acute pancreatitis	B) Biliary Disease	C) Alcohol abuse	D) Tropical pancreatitis
04 | Which of the following does not precipitate attacks of chronic pancreatitis			
A) Drugs such as opiates	B) Alcohol abuse	C) Overeating	D) Malabsorption
05 | which of the following is initially spared in chronic pancreatitis			
A) Acini	B) Ducts	C) Islets of Langerhans	D) Parenchyma
06 |Mutations in which of the following genes is associated with acute pancreatitis?			
A)PRSS1, SPINK1	B)p53	C)CFTR	D)NF2

MCQs Answer key	01	02	03	04	05	06
	B	B	C	D	C	A

QUIZ!

MCQs

07 | Primary acinar cell injury pathway is triggered by :			
A)Obstruction	B)Viral infection	C)Proenzyme activation	D) All of them
08 | Laboratory findings of acute pancreatitis in the 1st 24 hours include :			
A)Elevated serum amylase	B)Decreased serum amylase	C)Hypercalcemia	D)Decreased serum lipase
09 | The most common etiologies of acute pancreatitis (AP) are :			
A) Idiopathic and medications	B) Endoscopic retrograde cholangiopancreatography and gallstone	C) Abdominal trauma and pregnancy	D) Ethanol and gallstones
10 | The pancreatic ducts become extremely dilated in case of :			
A) Pseudocysts of Pancreas	B) Chronic Pancreatitis	C) Acute Pancreatitis	D) Autoimmune pancreatitis
11 | Which of the following is NOT a complication of chronic pancreatitis :			
A)Pancreatic exocrine insufficiency	B) Malabsorption	C) Acute ischemia	D) Diabetes mellitus
12 | A 54-year-old male alcoholic presents with the sudden onset of severe, constant epigastric pain that radiates to his mid back. Further evaluation finds fever, steatorrhea, and discoloration around his flank and umbilicus. Laboratory tests find elevated serum levels of amylase and lipase. What is the most likely cause of these findings :			
A)Acute appendicitis	B)Acute cholangitis	C)Acute diverticulitis	D)Acute pancreatitis
13 | A 45-year-old male presents with weight loss, steatorrhea, and malabsorption. A CT scan of the abdomen reveals a questionable mass in the head of the pancreas. A biopsy specimen microscopically reveals chronic inflammation and atrophy of the pancreatic acini with marked fibrosis. No malignancy is identified. What is the most common cause of this patient's disease in adults in the United States?			
A)Abdominal trauma	B)Cystic fibrosis	C)Chronic alcoholism	D)Hyperlipidemia

MCQs Answer key	07	08	09	10	11	12	13
	B	A	D	B	C	D	C

Pathoma

Acute and chronic pancreatitis

❖ ANNULAR PANCREAS

A. Developmental malformation in which the pancreas forms a ring around the duodenum; risk of duodenal obstruction

❖ ACUTE PANCREATITIS

A. Inflammation and hemorrhage of the pancreas
B. Due to autodigestion of pancreatic parenchyma by pancreatic enzymes
1. Premature activation of trypsin leads to activation of other pancreatic enzymes.
C. Results in liquefactive hemorrhagic necrosis of the pancreas and fat necrosis of the peripancreatic
D. Most commonly due to alcohol and gallstones; other causes include trauma, hypercalcemia, hyperlipidemia, drugs, scorpion stings, mumps, and rupture of a posterior duodenal ulcer.
E. Clinical features
1. Epigastric abdominal pain that radiates to the back
2. nausea and vomiting
3. Periumbilical and flank hemorrhage (necrosis spreads into the periumbilical soft tissue and retroperitoneum)
4. Elevated serum lipase and amylase; lipase is more specific for pancreatic damage.
5. Hypocalcemia (calcium is consumed during saponification in fat necrosis)
F. Complications
l. Shock- due to peripancreatic hemorrhage and fluid sequestration
2. Pancreatic pseudocyst-formed by fibrous tissue surrounding liquefactive necrosis and pancreatic enzymes
i. Presents as an abdominal mass with persistently elevated serum amylase
ii. Rupture is associated with release of enzymes into the abdominal cavity and hemorrhage.
3. Pancreatic abscess-often due to E coli; presents with abdominal pain, high fever, and persistently elevated amylase
4. DIC and ARDS

❖ CHRONIC PANCREATITIS

A. Fibrosis of pancreatic parenchyma, most often secondary to recurrent acute pancreatitis
1. Most commonly due to alcohol (adults) and cystic fibrosis (children); however, many cases are idiopathic.
B. Clinical features:
l. Epigastric abdominal pain that radiates to the back
2. Pancreatic insufficiency-results in malabsorption with steatorrhea and fat soluble vitamin deficiencies. Amylase and lipase are not useful serologic markers of chronic pancreatitis.
3. Dystrophic calcification of pancreatic parenchyma on imaging; contrast studies reveal a 'chain of lakes' pattern due to dilatation of pancreatic ducts.
4. Secondary diabetes mellitus-late complication due to destruction of islets
5. Increased risk for pancreatic carcinoma

" اللهم علمنا ماينفعنا ، وانفعنا بما علمتنا وزدنا علما يارب العالمين "

Team leaders

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- Hassan alshurafa
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- Raghad Alassiry
- Fahad Alajmi
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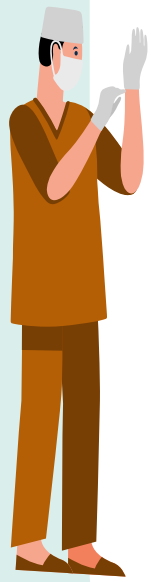

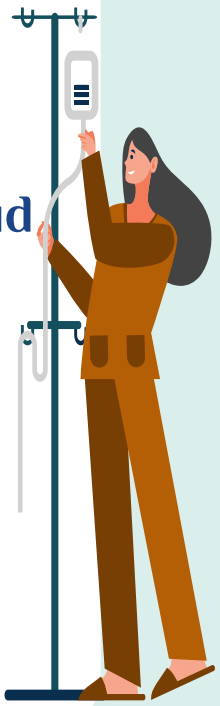

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- Member
- Note taker
- Reviser

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