Acute and chronic pancreatitis

By Josefine Holum



- Physiology of the pancreas
- Acute pancreatitis
- Chronic pancreatitis



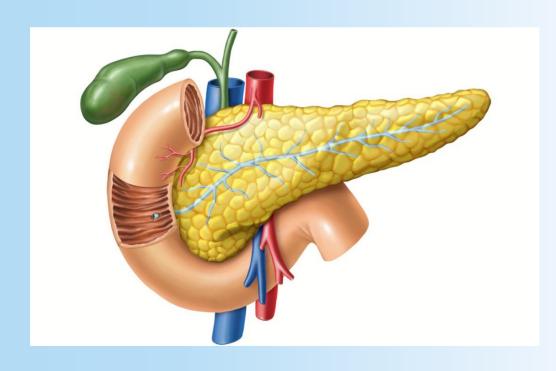
Pancreas

Endocrine function

- Alpha cells (glucagon)
- Beta cells (insulin)
- Delta cells (somatostatin)
- PP cells (pancreatic popypeptide)

Exocrine function

- Production of digestive enzymes
- Amylase, lipase, protease, trypsin



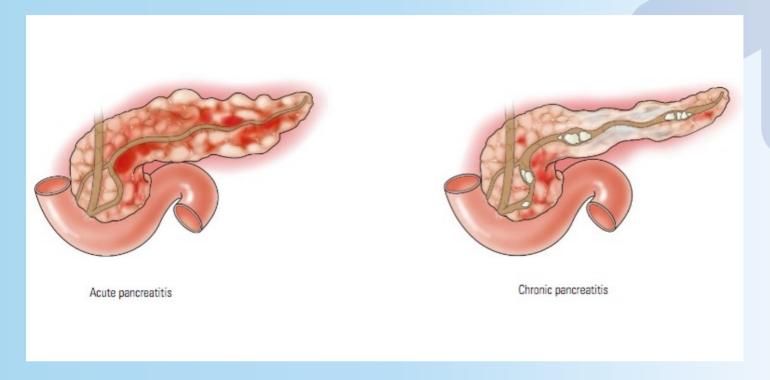


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Pancreatitis

- Inflammation of the pancreas
- Acute inflammation
- Chronic inflammation



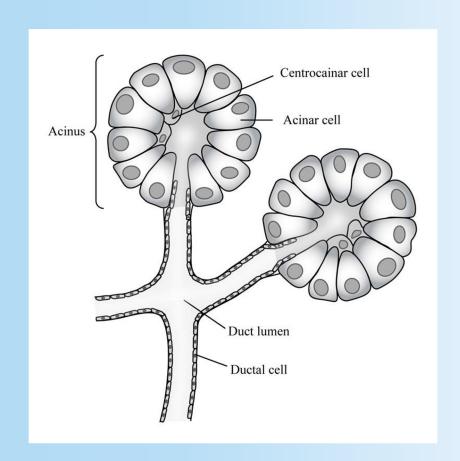


Acinar cells

Produce digestive enzymes

Ductal cells

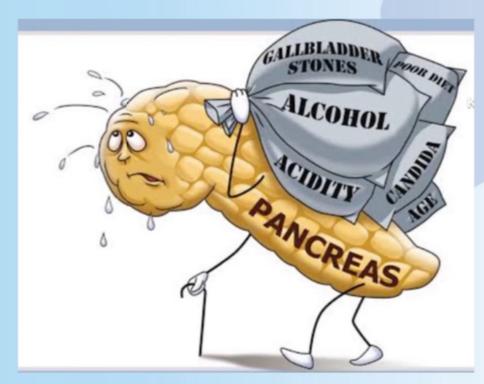
• Form the epithelial lining of the pancreatic ducts





Causes of acute pancreatitis

- I Idiopathic
- **G** Gallstones
- E Ethanol abuse
- T Trauma
- S Steroids
- M Mumps virus
- A Autoimmune disorders
- S Scorpion stings
- H Hypertriglyceridemia and hypercalcemia
- E ERCP
- D Drugs







Alcohol <3

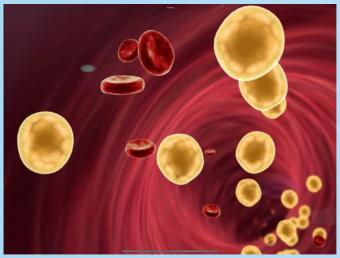




Alcohol <3

Hypertriglyceridemia





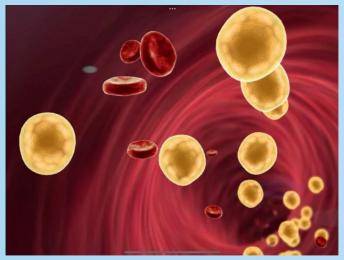


Alcohol <3

Hypertriglyceridemia

Drugs

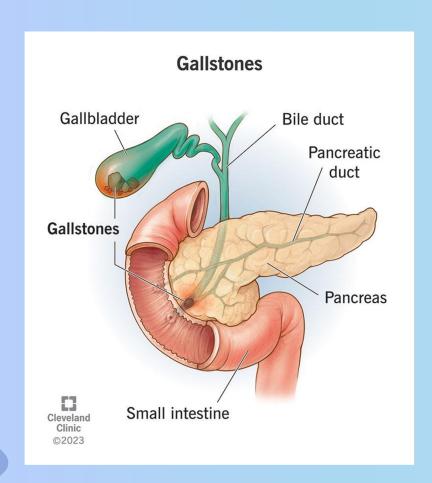






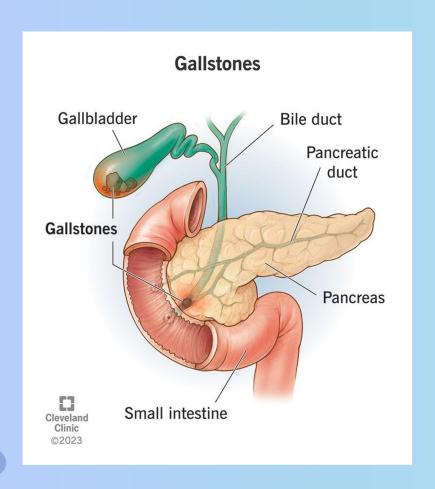


Ductal cell damage

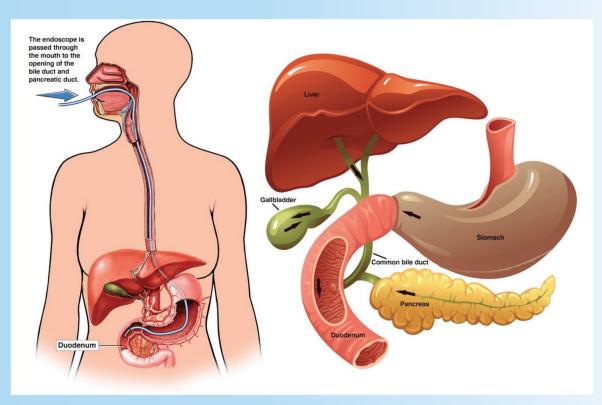




Ductal cell damage



ERCP





Refresher

1. Inflammation

1. Recruitment of white blood cells

1. Increased vascular permeability



Fluid spacing

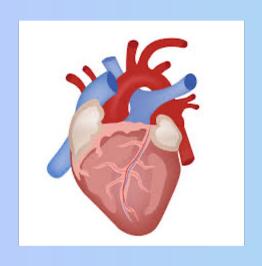
First spacing: All fluids are where they should be. There is normal distribution of fluid in the intra- and extracellular fluid.

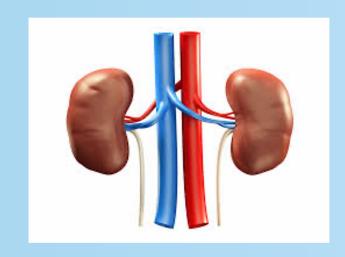
Second spacing: Abnormal accumulation of fluid in interstitial spaces, such as edema. This abnormal accumulation can still be easily moved back where it should be

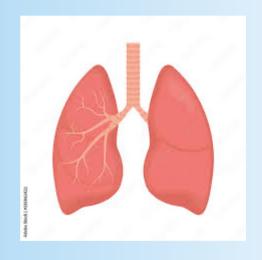
Third spacing: Abnormal accumulation of fluid trapped in spaces where it is difficult or impossible to return to where it should be such as ascites or burn related injuries. This requires medical intervention to reverse



Systemic inflammatory response syndrome







- Increased heart rate
- Low BP

- Low kidney perfusion
- AKI

- Diffuse alveolar damage
- ARDS



Acinar cell injury lead to:

(1) Interstitial inflammation and edema

(2) Proteolysis

Action of proteases: increased vascular permeability Blood vessels leak and rupture → ARDS

(3) Fat necrosis

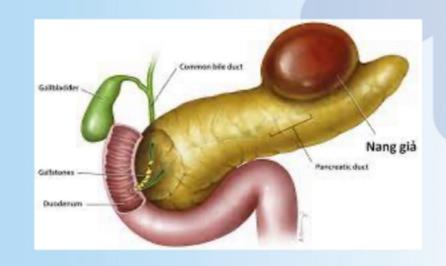
Action of lipase and phospholipase Destruction of peripancreatic fat

(4) Hemorrhage

→ Hypovolemia & septic shock

Action of elastase: destroys elastic tissue of blood vessels

Digestion and bleeding can liquefy tissue: liquefactive hemorrhagic necrosis and pancreatic pseudocyst



Diagnostic picture

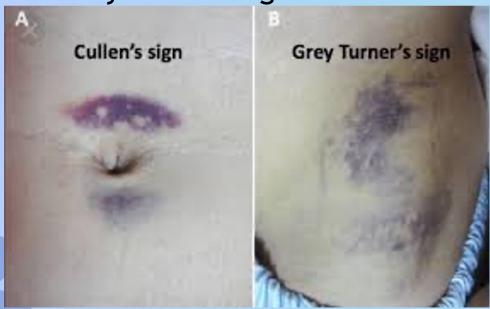
- Epigastric pain
- Pain radiates to the back
- Abdominal tenderness
- Cullen's sign
- Grey Turner sign

Lab values:	Explanation:
∏ Lipase	Rise within 8 hours
(3x normal	Return to normal within 14 days
value)	Autodigestion of pancreas (consequence of
	acute pancreatitis) results in release of lipase
	and amylase.
↑ Amylase	Rise within 12 hours
(3x normal	Return to normal within 5 days
value)	Autodigestion of pancreas (consequence of
	acute pancreatitis) results in release of lipase
	and amylase.
↑ Leukocytes	Increased due to dehydration or hemorrhaging
& hematocrit	
介 CRP and	Increased due to inflammation
LDH	
↑ BUN &	Renal insufficiency, pancreatic necrosis and
Creatinine	dehydration
↓ Calcium	Decreased due to that fat necrosis consume
	Ca ²⁺

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Chronic pancreatitis

- (1) Repeated bounces of acute pancreatitis
- (2) Persistent inflammation cause changes in structure

Ductal dilation

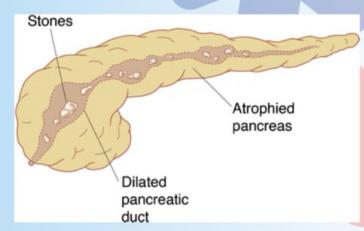
Stellate cells produce fibrotic tissue causing stenosis

Calcium deposition → Plugs



Destruction of pancreatic b-cells → Diabetes mellitus

Acinar cell atrophy → Decreased production of digestive enzymes → ADEK deficiency & Steatorrhea







Alcohol abuse





Ductal cell damage

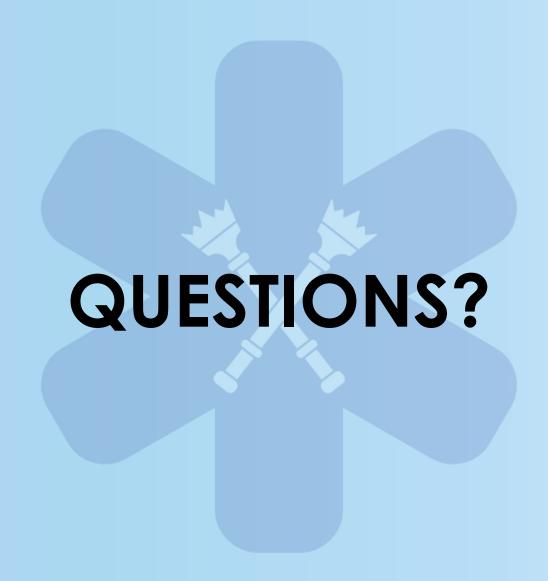
Alcohol abuse



Cystic fibrosis









Thank you lovelies <3

