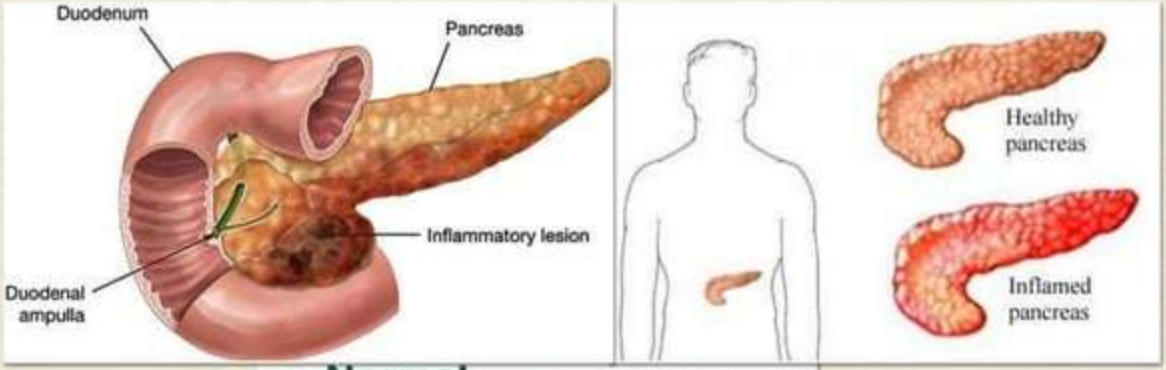
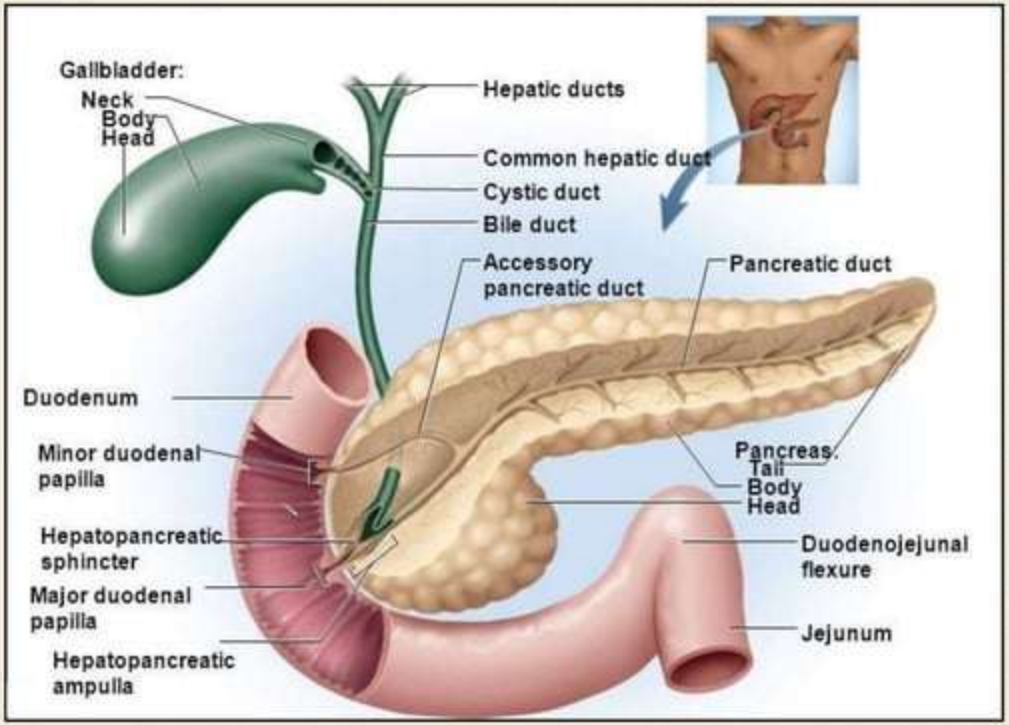
PANCREATITIS

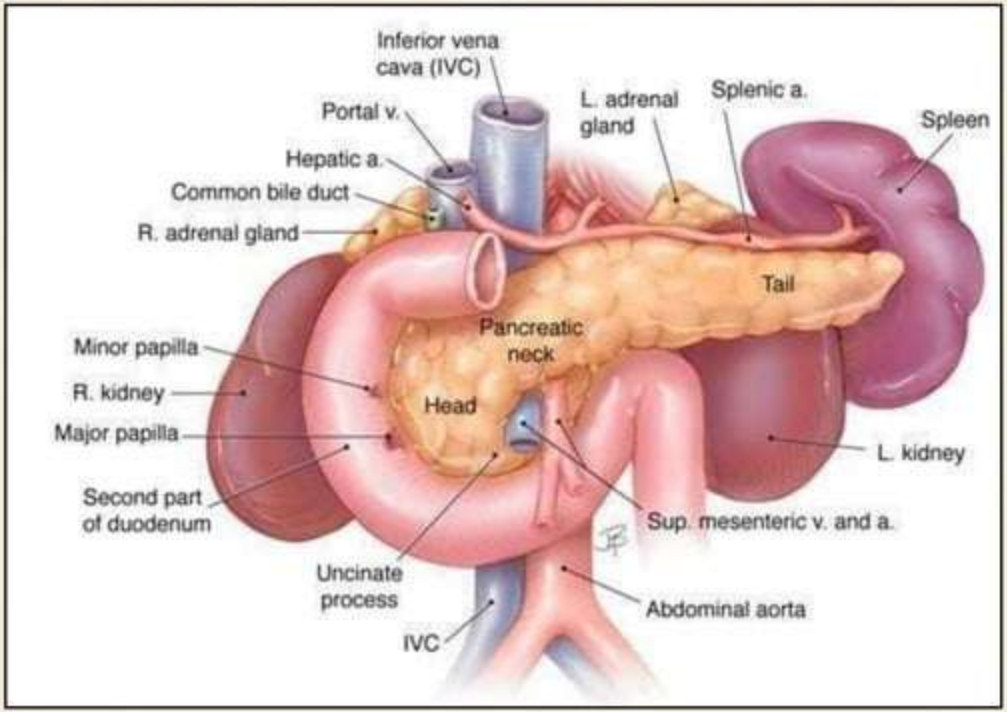


By : Ankita Priydarshini
Roll No. - 21

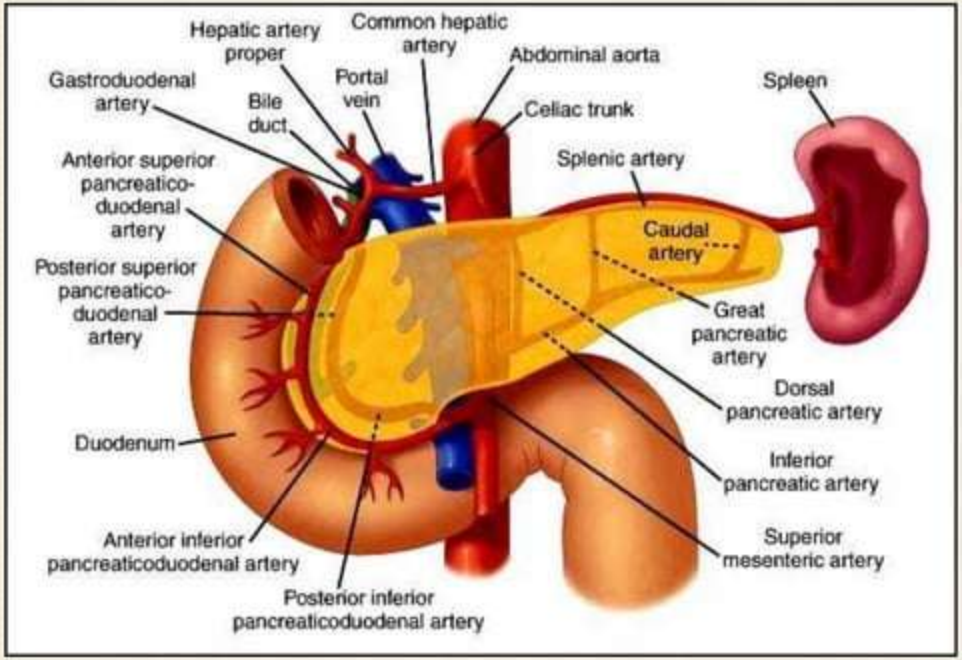
ANATOMY


Gallbladder:
Neck
Body
Head
Hepatic ducts
Common hepatic duct
Cystic duct
Bile duct
Accessory pancreatic duct
Pancreatic duct
Duodenum
Minor duodenal papilla
Pancreas:
Tail
Body
Head
Hepatopancreatic sphincter
Duodenojejunal flexure
Major duodenal papilla
Jejunum
Hepatopancreatic ampulla

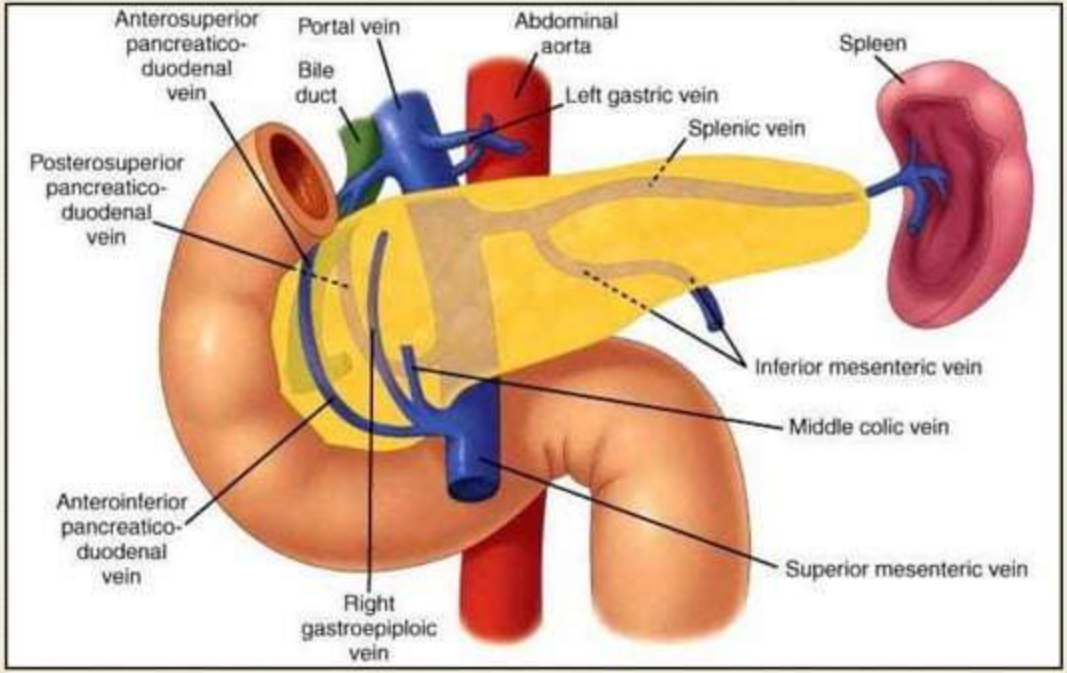
RELATIONS OF PANCREAS



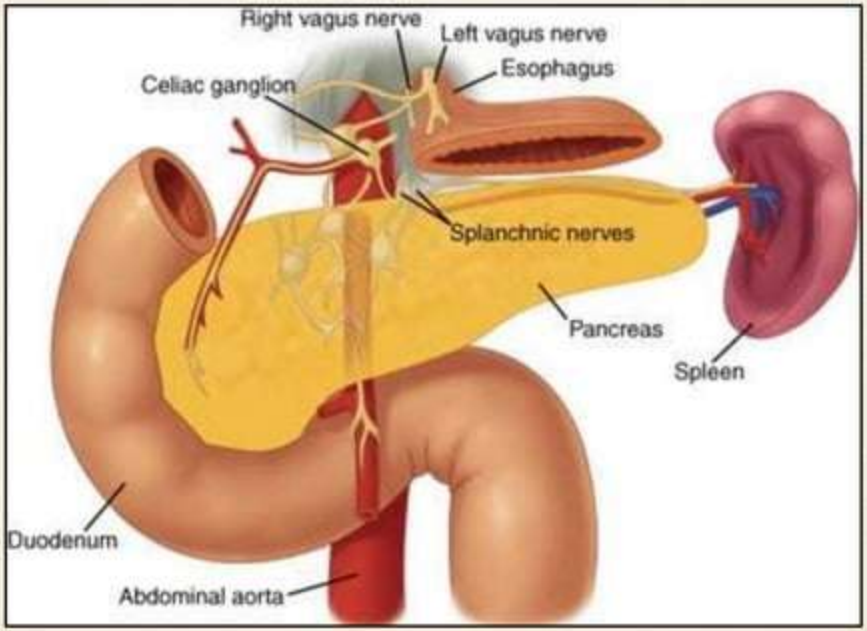
ARTERIAL SUPPLY



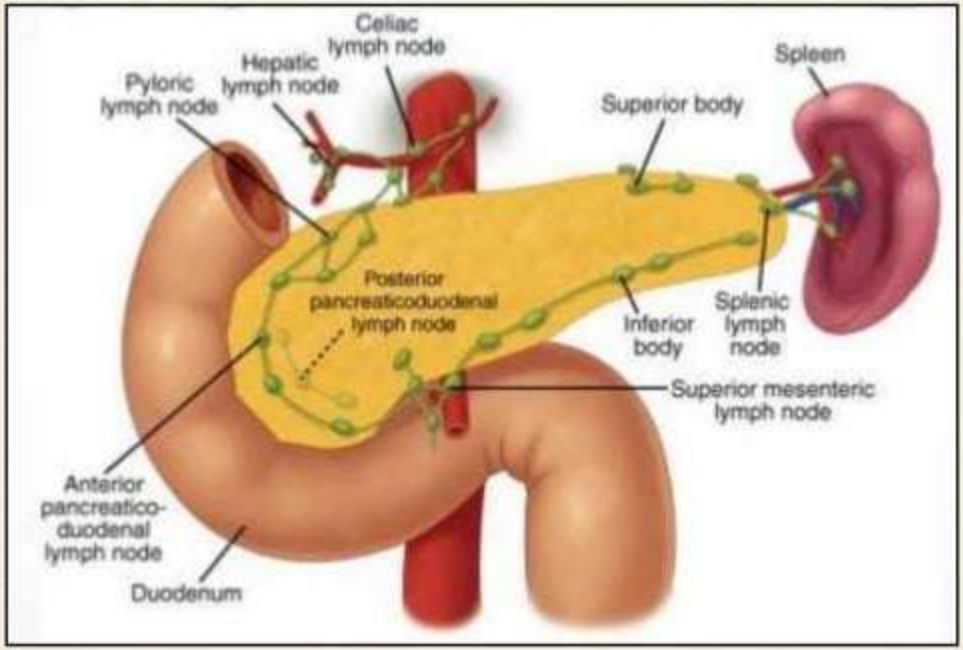
VENOUS DRAINAGE



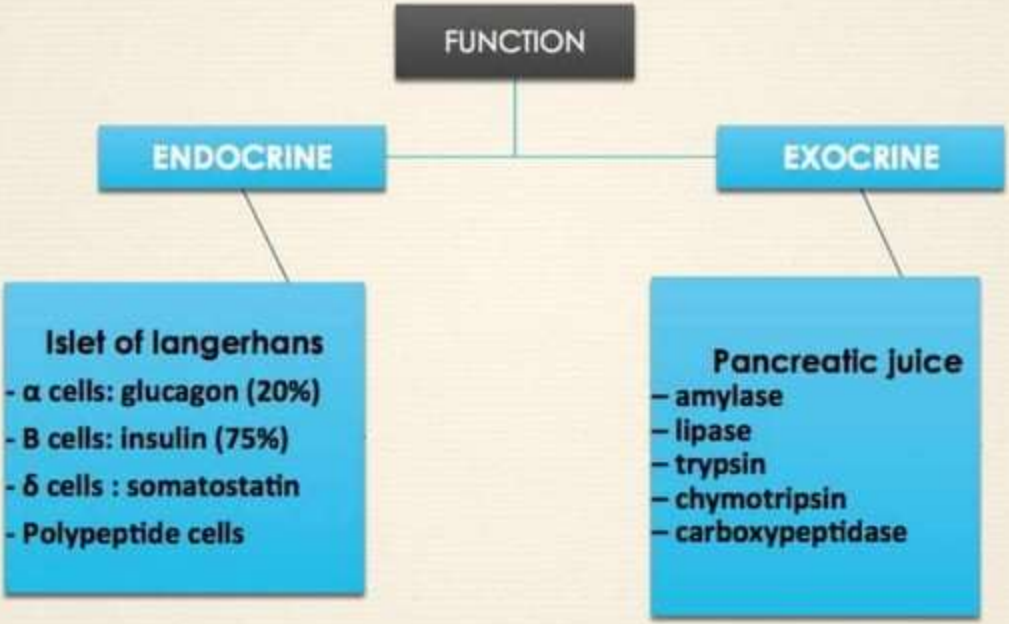
NERVE SUPPLY

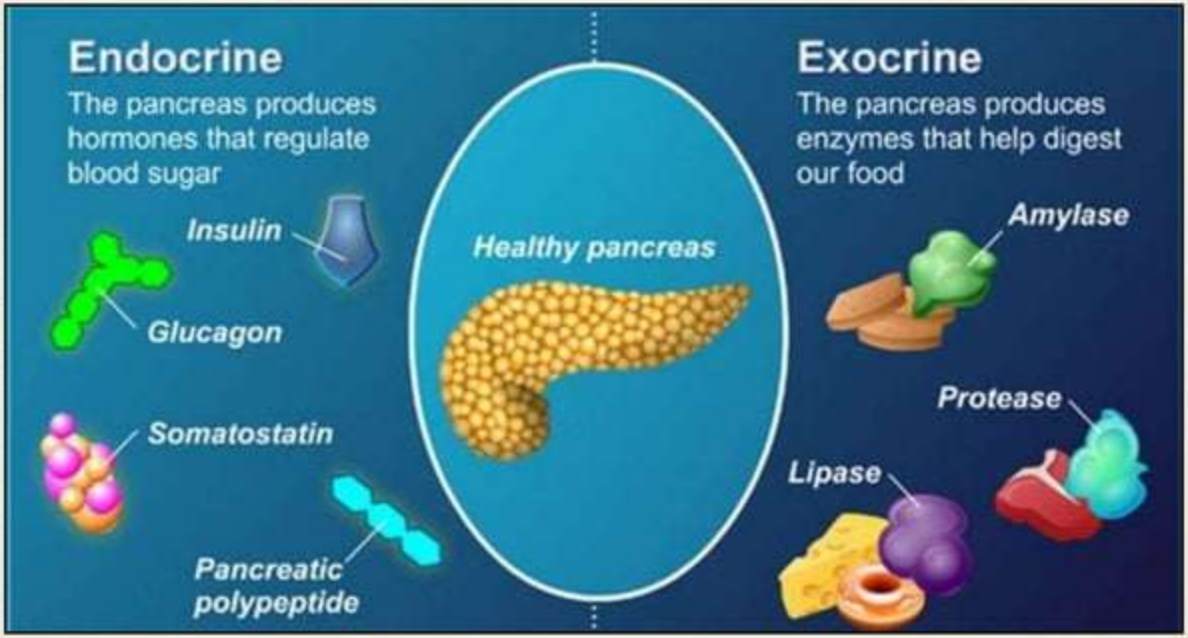


LYMPHATIC DRAINAGE

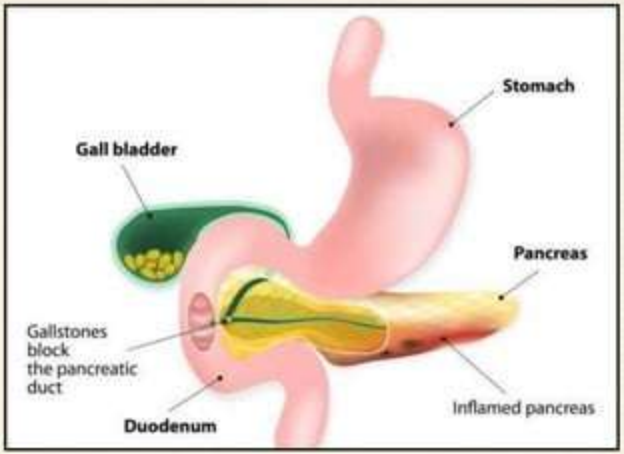


PHYSIOLOGY


FUNCTION
ENDOCRINE
EXOCRINE
Islet of langerhans
- α cells: glucagon (20%)
- B cells: insulin (75%)
- δ cells : somatostatin
- Polypeptide cells
Pancreatic juice
– amylase
– lipase
– trypsin
– chymotripsin
– carboxypeptidase


Endocrine
The pancreas produces hormones that regulate blood sugar
Insulin
Glucagon
Somatostatin
Pancreatic polypeptide
Healthy pancreas
Exocrine
The pancreas produces enzymes that help digest our food
Amylase
Protease
Lipase

ACUTE PANCREATITIS



- A group of reversible lesions characterised by inflammation of the pancreas.
- Acute pancreatitis is a condition in which activated pancreatic enzymes leak into the substance of the pancreas and initiate the auto-digestion of the gland.

Etiology

- **Non-traumatic(75%)**

Biliary tract diseases

Alcohol

Viral infection (EBV, CMV, mumps)

Drugs (steroid, thiazide, furosemide)

Scorpion bites

Metabolic (Hyperlipidemia, Hypertriglyceridemia)

Causes of **Pancreatitis**: (**BAD HITS**)

- **B**iliary
- **A**lcohol
- **D**rugs (Corticosteroids, HIV drugs, Diuretics, Valproic acid…)
- **H**ypertriglyceridemia/**H**ypercalcemia
- **I**diopathic
- **T**rauma
- **S**corpion sting

- **Traumatic (5%)**

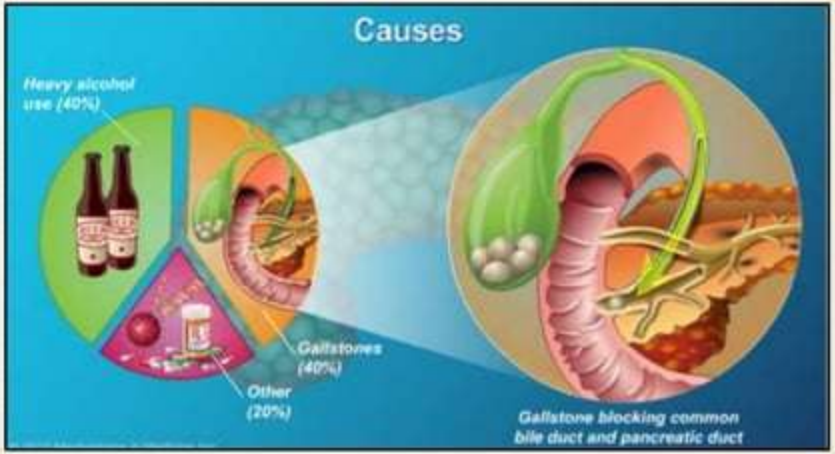
Operative trauma

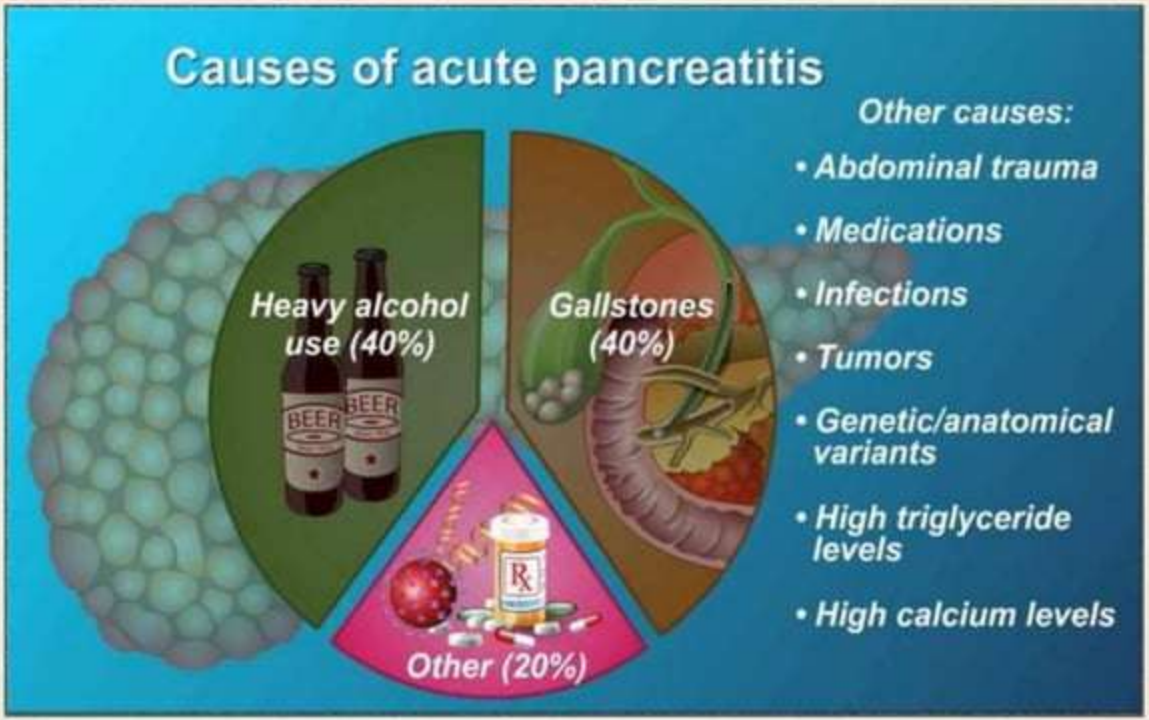
Blunt/penetrating trauma

Post - ERCP (back pressure of contrast)

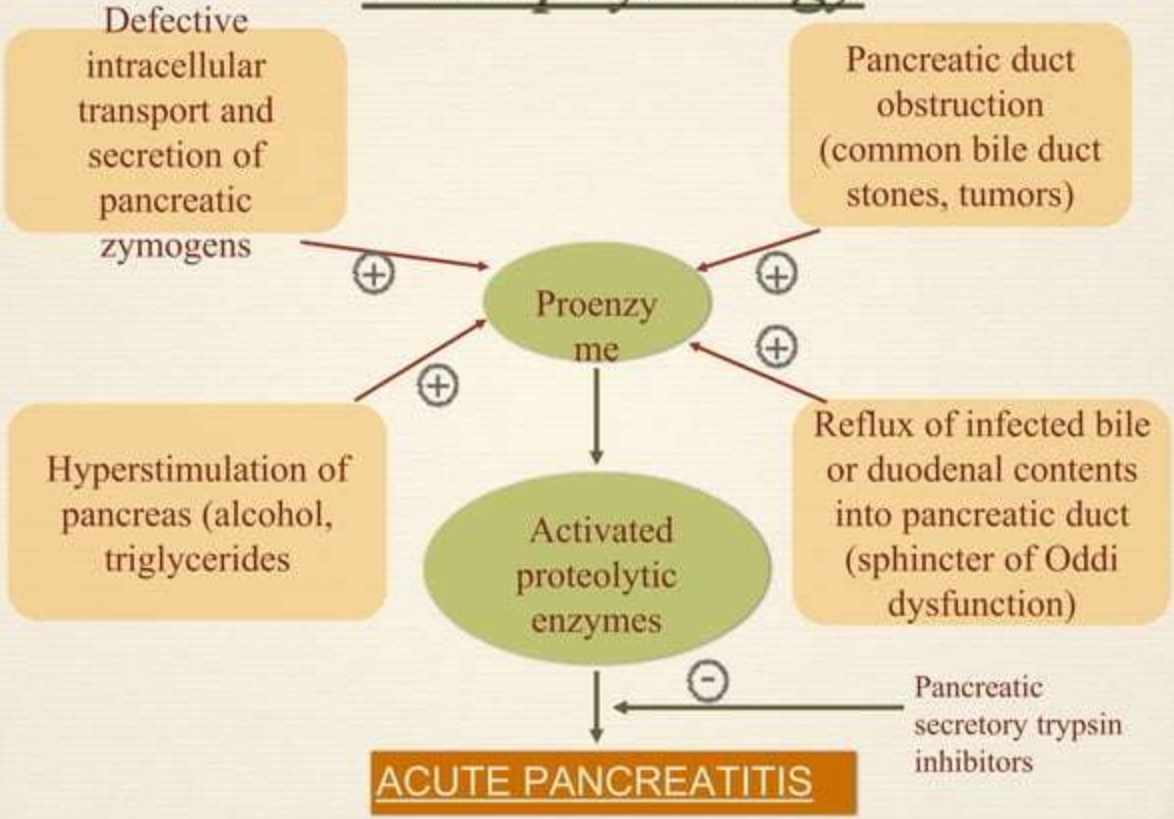
- **Idiopathic(20%)**

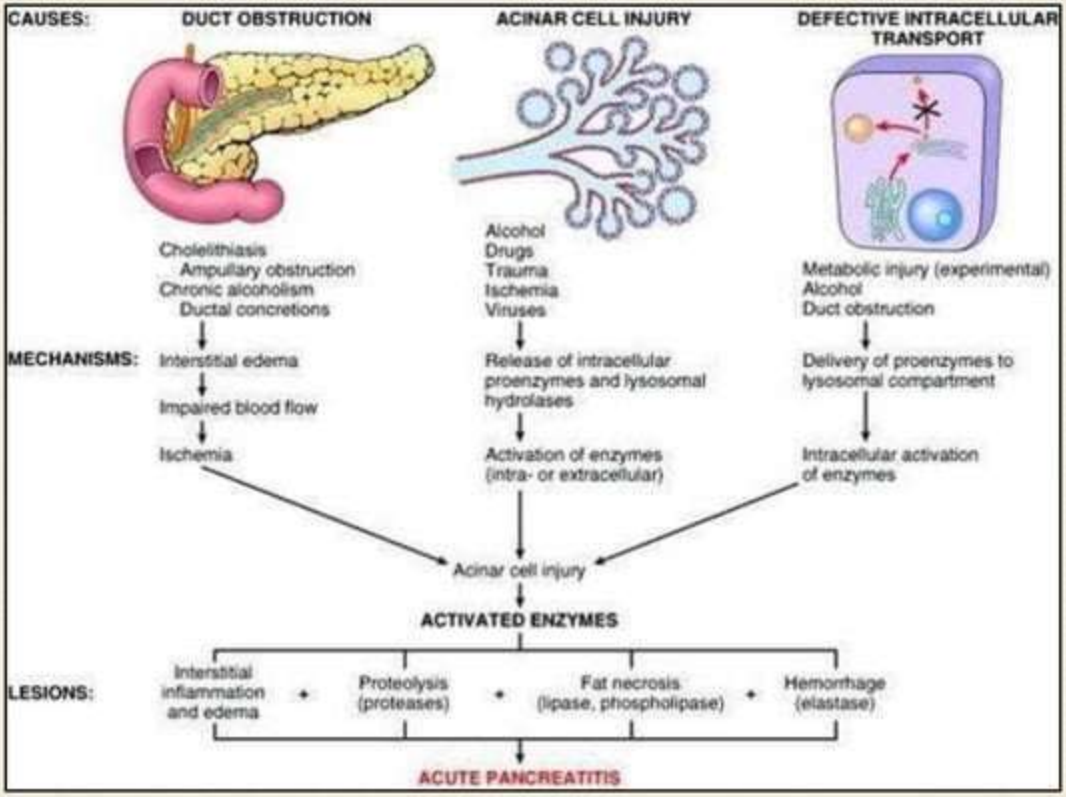

Alcohol abuse and gallstones
are the main triggers of acute pancreatitis
BEER
BEER
Alcohol
abuse
Gallstones

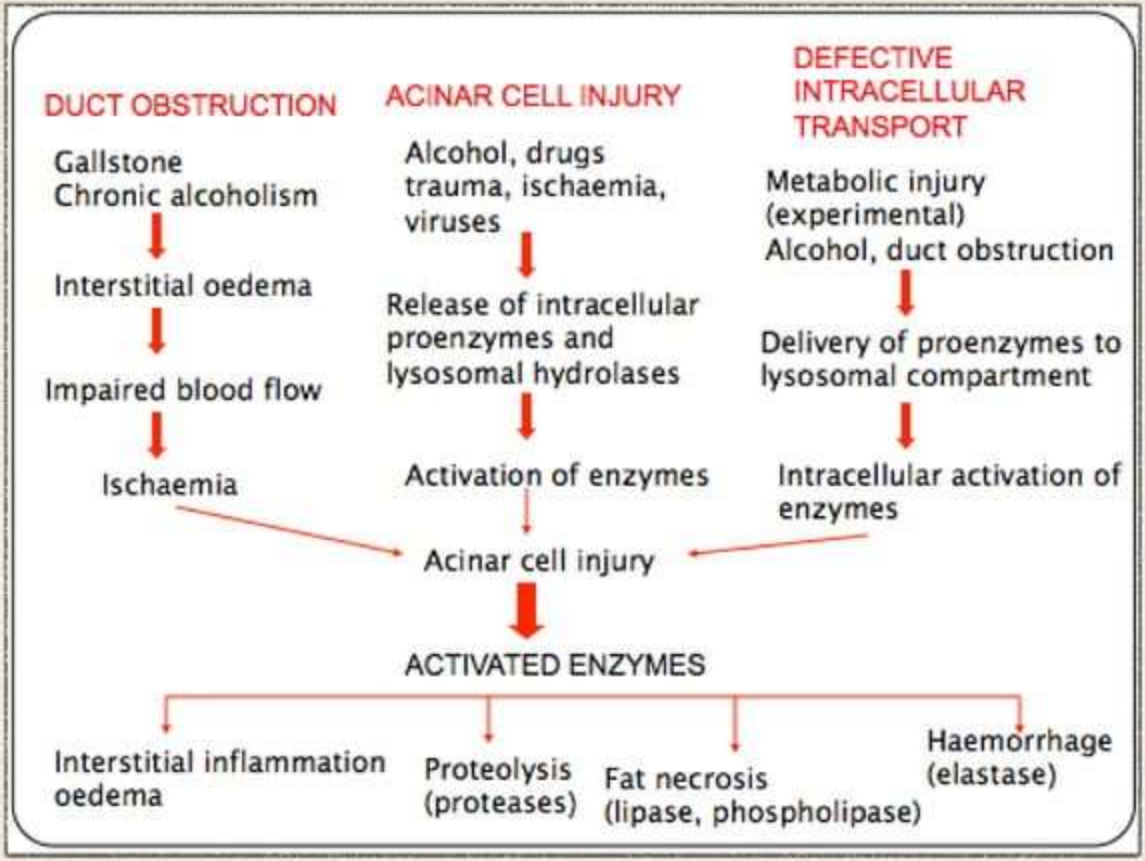

Causes
Heavy alcohol
use (40%)
Gallstones
(40%)
Other
(20%)
Gallstone blocking common
bile duct and pancreatic duct


Causes of acute pancreatitis
Heavy alcohol use (40%)
BEER
BEER
Gallstones (40%)
Rx
Other (20%)
Other causes:
• Abdominal trauma
• Medications
• Infections
• Tumors
• Genetic/anatomical variants
• High triglyceride levels
• High calcium levels

Pathophysiology




CAUSES:
DUCT OBSTRUCTION
ACINAR CELL INJURY
DEFECTIVE INTRACELLULAR TRANSPORT
Cholelithiasis
Ampullary obstruction
Chronic alcoholism
Ductal concretions
Alcohol
Drugs
Trauma
Ischemia
Viruses
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


DUCT OBSTRUCTION
Gallstone
Chronic alcoholism
Interstitial oedema
Impaired blood flow
Ischaemia
ACINAR CELL INJURY
Alcohol, drugs
trauma, ischaemia,
viruses
Release of intracellular
proenzymes and
lysosomal hydrolases
Activation of enzymes
DEFECTIVE
INTRACELLULAR
TRANSPORT
Metabolic injury
(experimental)
Alcohol, duct obstruction
Delivery of proenzymes to
lysosomal compartment
Intracellular activation of
enzymes
Acinar cell injury
ACTIVATED ENZYMES
Interstitial inflammation
oedema
Proteolysis
(proteases)
Fat necrosis
(lipase, phospholipase)
Haemorrhage
(elastase)

Acute Pancreatitis

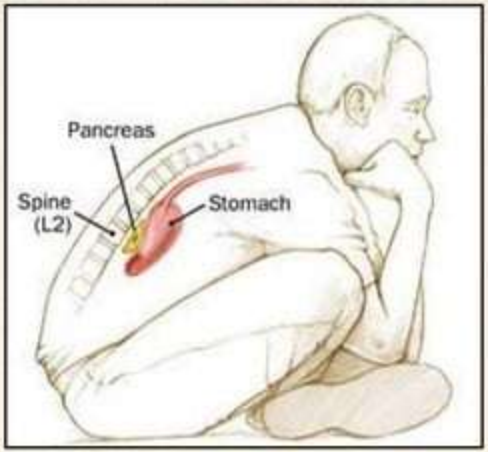
Two phases	**Early**	**Late**	
	1st week	After 1st week	
Severity	**Mild**	**Moderate**	**Severe**
	No organ failure	Organ failure less than 48 h	Organ failure longer than 48 h
Two types	**Oedematous**	**Necrotizing**	
Complications	< 4 wk : acute peripancreatic collection > 4 wk: pseudocyst	< 4 wk : acute necrotic collection > 4 wk : walled-off necrosis	

Clinical presentation

- **Mid-epigastric pain** with tenderness. Sudden severe pain occurring often within 12-24 hours of a large meal or alcohol. The pain is persistent and radiates frequently through to the back to either shoulder or to one iliac fossa before spreading to involve the whole abdomen. Exacerbated on walking and lying supine. Relieved on sitting and leaning forward.

- **Nausea and vomiting** has always been the presentation of acute pancreatitis in the majority of cases.

- When pancreatitis is extremely severe, it mimics septic **shock;** fever, hypotension, respiratory distress from ARDS, elevation of the WBC and a rigid abdomen.


Pancreas
Spine
(L2)
Stomach


Prematurely activated digestive enzymes
cause inflammation of the pancreas
Intense pain
Autodigestion of the pancreas
leads to inflammation
Vomiting

Abdominal Presentation

- Tenderness in epigastrium
- Although severe pain, there may be little or no guarding of abdominal muscles at first. Later the upper abdomen becomes tender and rigid as peritoneal irritation increases.
- Mild abdominal distention if paralytic ileus develops.
- Severe advanced cases may develop bruising and discoloration in the left flank (**Grey Turner's sign** due to tissue catabolism of Hb) and around the umbilicus (**Cullen's sign** due to hemoperitoneum). These are the rare and late signs of extensive pancreatic destruction.

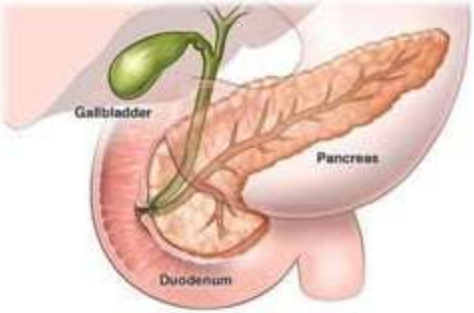
Cullen's Sign

Grey Turner's Sign

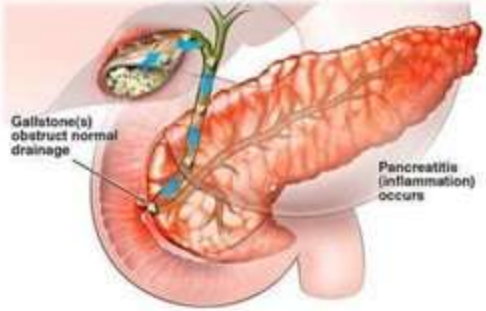
Complications of Acute Pancreatitis

Complications	Causes and features
Shock and renal failure	Pancreatic failure is associated with leakage of fluid in the pancreatic bed also ileus with fluid filled loops of bowel leading to pre-renal azotemia and then acute tubular necrosis.
Hypoxia	ARDS due to micro thrombi in pulmonary vessels.
Hyperglycemia	Due to disruption of pancreatic islets.
Hypocalcemia	Sequestration of calcium in fat necrosis.
Hypoalbuminemia	Increased capillary permeability.

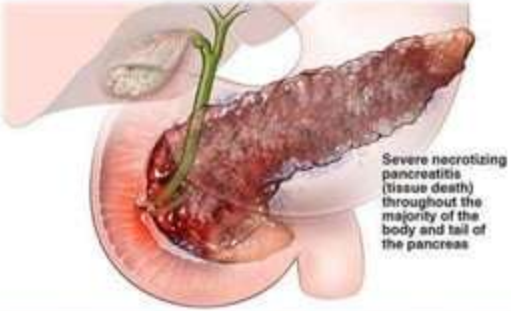
Normal Gallbladder and Pancreas



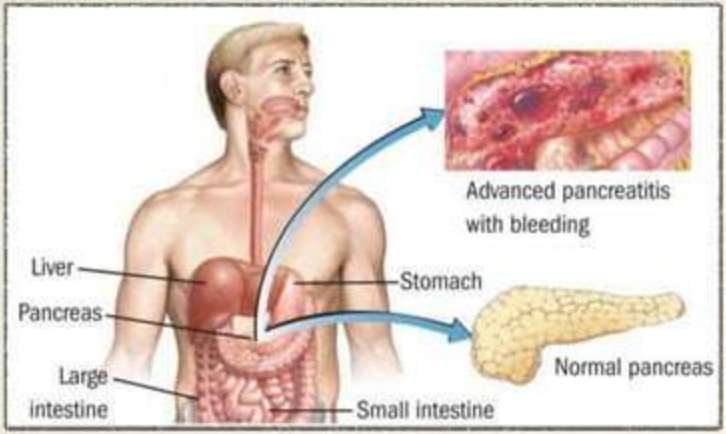
Pancreatitis

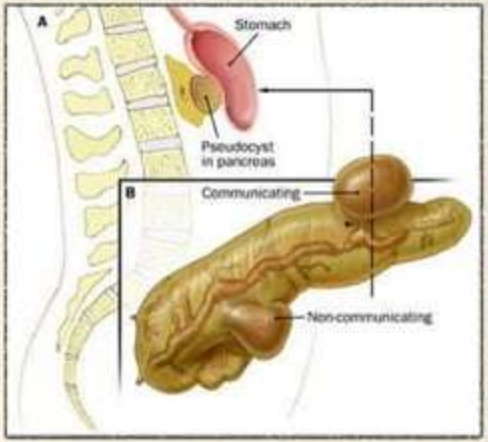


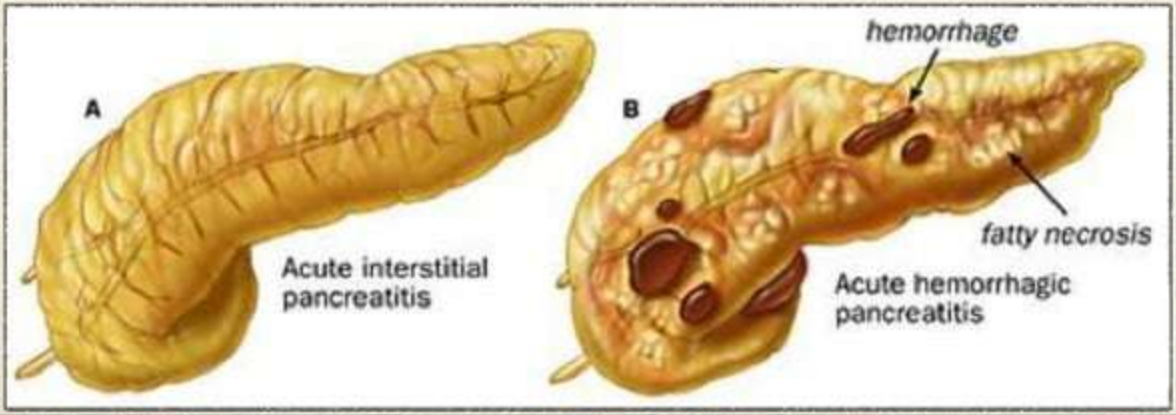
Severe Necrotizing Pancreatitis



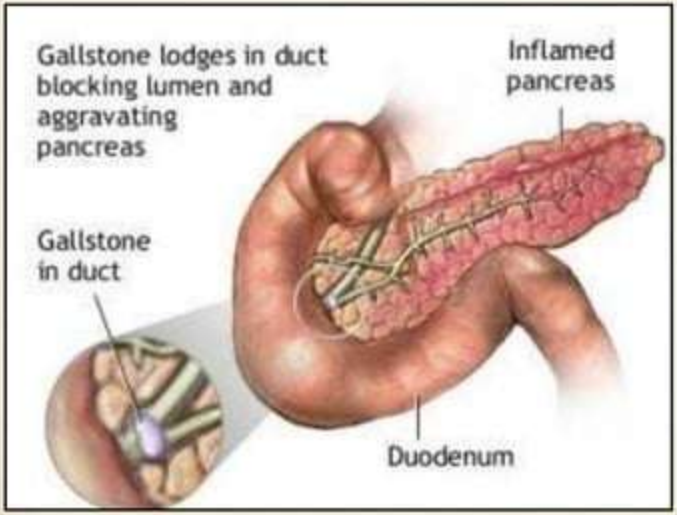
Pancreatic complications	Causes and features
Necrosis	
Abscess	Rising fever, leukocytosis, localized tenderness and epigastric mass. It may be associated with left sided pleural effusion and enlarged spleen due to splenic vein thrombosis.
Pseudocyst	Encapsulated fluid collection with high enzyme content. Usually less than 6cm sized pseudocysts resolve spontaneously. They may become secondarily infected requiring drainage of abscess.
Ascites	Gradual increase in abdominal girth and persistent elevation of serum amylase in the absence of frank abdominal pain. It results from rupture of pancreatic duct or drainage of pseudocyst into the pancreatic cavity.

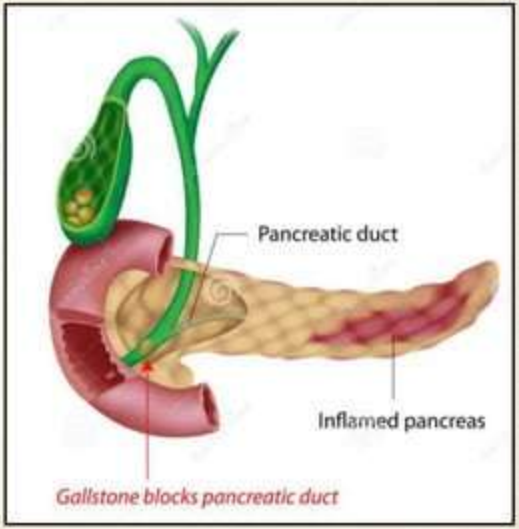

Advanced pancreatitis
with bleeding
Liver
Stomach
Pancreas
Normal pancreas
Large
intestine
Small intestine


A
Stomach
Pseudocyst
in pancreas
B
Communicating
Non-communicating


A
Acute interstitial
pancreatitis
B
hemorrhage
fatty necrosis
Acute hemorrhagic
pancreatitis

Gastrointestinal complications	Causes and features
Upper GI bleeding	Gastric or duodenal erosion
Duodenal obstruction	Compression by pancreatic mass
Obstructive jaundice	Compression of common bile duct





Pancreatitis

Inflammation of the parenchyma of the pancreas

Acute

Presents as an acute abdomen condition

Chronic

- Prolonged & frequently lifelong disorder.
- Development of fibrosis within the pancreas


Eventually leads to irreversible damage of pancreas
Destruction of
pancreatic tissue

CHRONIC PANCREATITIS

It is defined as permanent & irreversible damage to pancreas with histologic evidence of chronic inflammation, fibrosis & destruction of exocrine (acinar cells) & endocrine (islets of Langerhans) tissues.

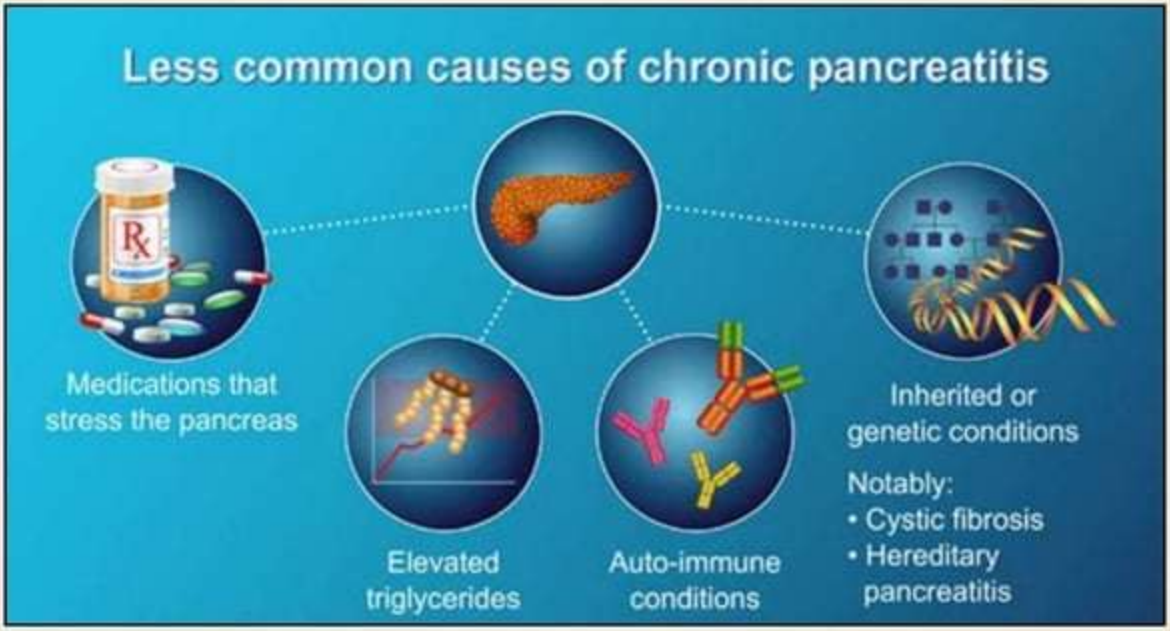
- **The chief distinction between acute and chronic pancreatitis is the**

irreversible

impairment in pancreatic function that is characteristic of chronic pancreatitis.

Etiology


Causes of chronic pancreatitis
Lifestyle factors in predisposed patients
Long-standing, heavy alcohol use (although not always the case)
BEER
BEER
Long-term heavy smoking
Chronic pancreatitis develops slowly over time


Less common causes of chronic pancreatitis
Medications that stress the pancreas
Elevated triglycerides
Auto-immune conditions
Inherited or genetic conditions
Notably:
• Cystic fibrosis
• Hereditary pancreatitis

TIGAR-O Risk Factor

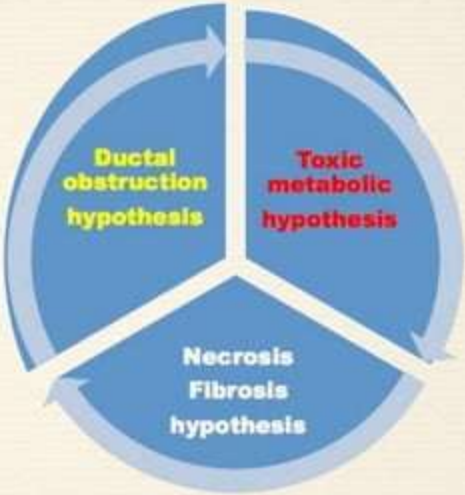
- T - Toxic - alcohol/tobacco/dietary/drug
- I- Idiopathic
- G- Genetic Mutations -CFTR/SPINK1
- A- Autoimmune primary with Sjogren/Crohn's disease
- R- Recurrent & severe acute / ischemia
- O- Obstructive - annular pancreas/stenotic papilla/ duodenal obstruction/trauma/pancreatic duct stones

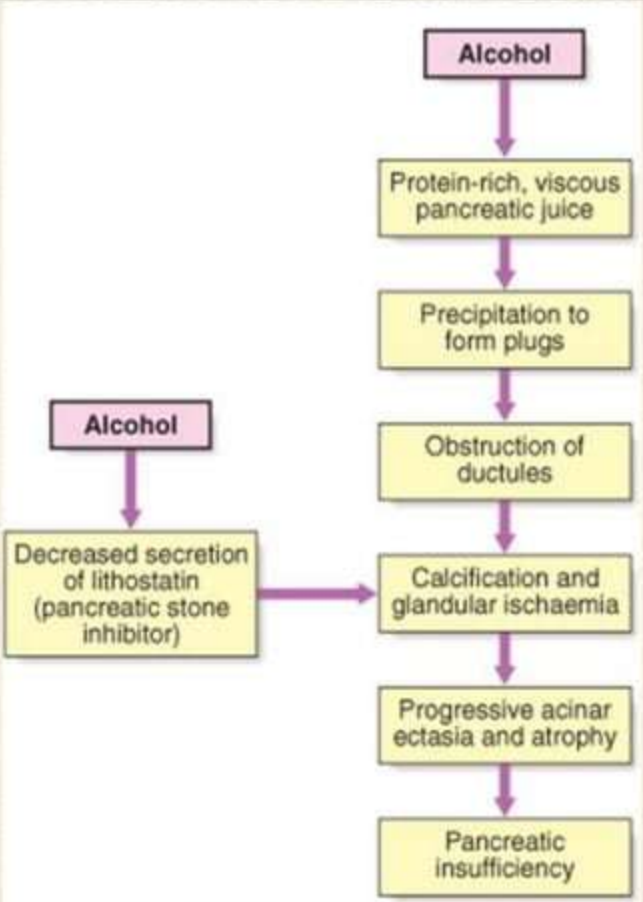
Pathophysiology

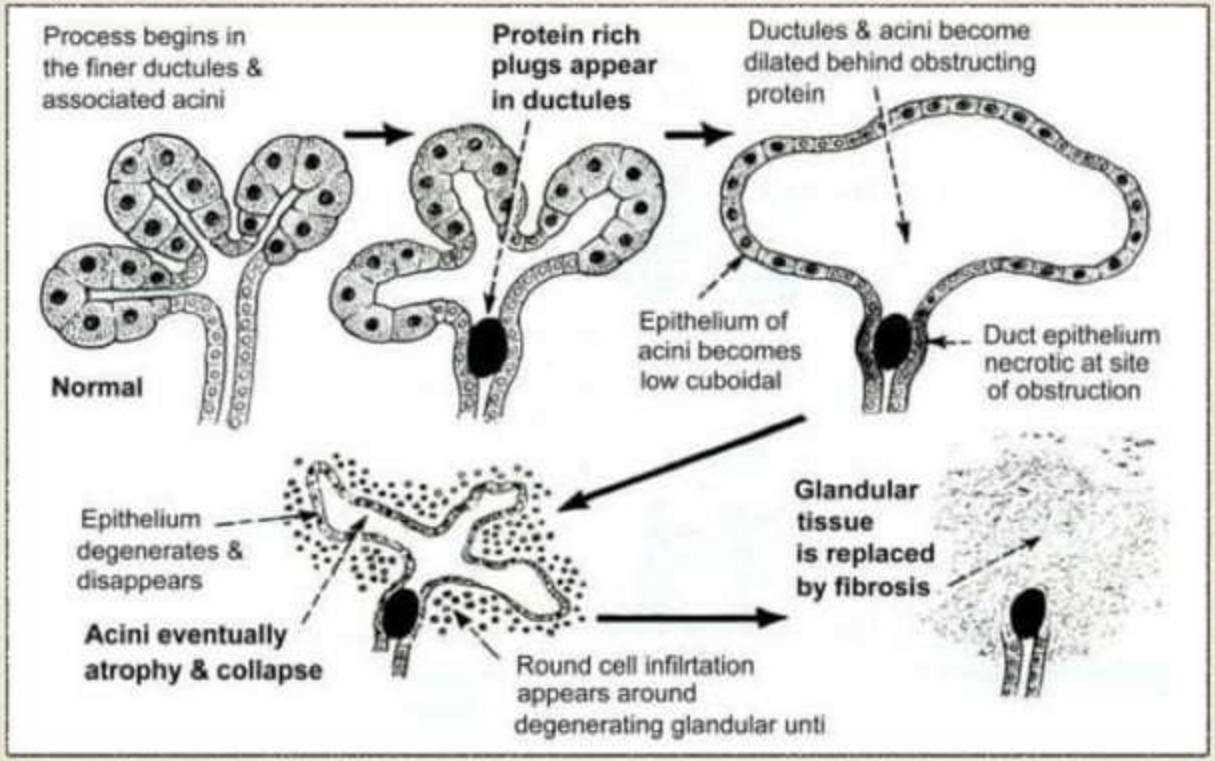
Not well understood

Almost all individuals with repeated episodes of acute pancreatitis later develop chronic pancreatitis.

Sentinel Accute Pancreatitis Events (SAPE) Hypothesis



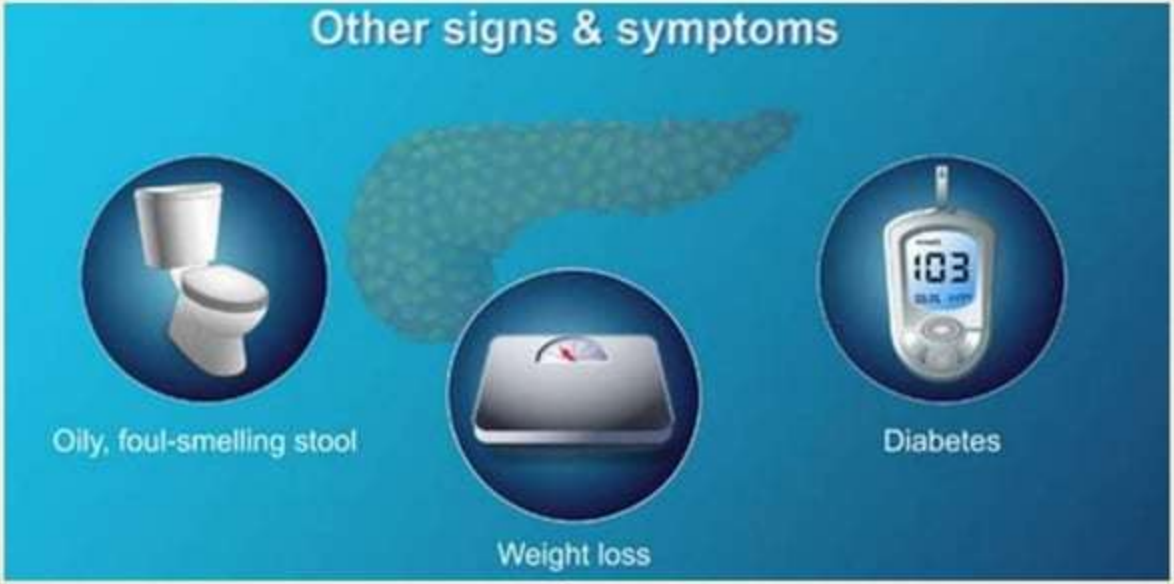

Alcohol
Protein-rich, viscous pancreatic juice
Precipitation to form plugs
Alcohol
Obstruction of ductules
Decreased secretion of lithostatin (pancreatic stone inhibitor)
Calcification and glandular ischaemia
Progressive acinar ectasia and atrophy
Pancreatic insufficiency


Process begins in the finer ductules & associated acini
Protein rich plugs appear in ductules
Ductules & acini become dilated behind obstructing protein
Normal
Epithelium of acini becomes low cuboidal
Duct epithelium necrotic at site of obstruction
Epithelium degenerates & disappears
Acini eventually atrophy & collapse
Round cell infilrtation appears around degenerating glandular unti
Glandular tissue is replaced by fibrosis

Clinical presentation

- Type -A : pain-free intervals
- Type -B : unrelenting pain

- Mallet Guys Sign - In right knee chest position, if left hypochondrium is palpated tenderness can be elicited.



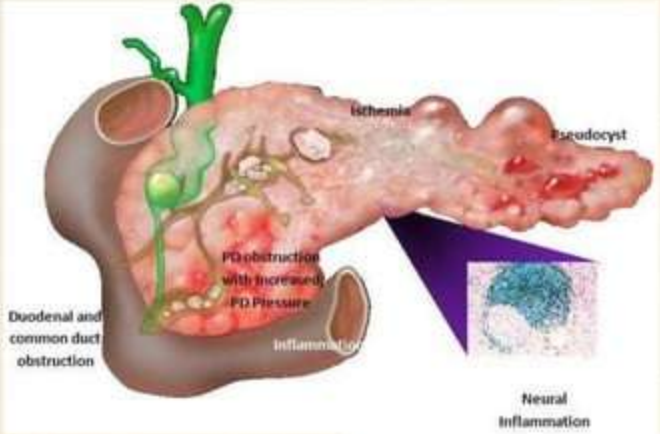
Triad of Chronic Pancreatitis

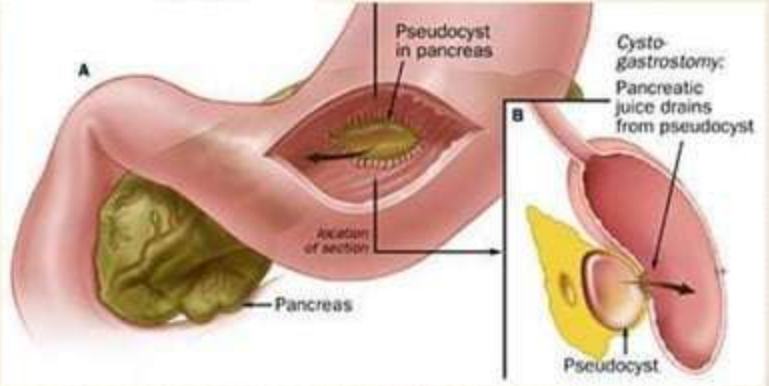
- Pancreatic calcification
- Steatorrhoea
- Diabetes mellitus

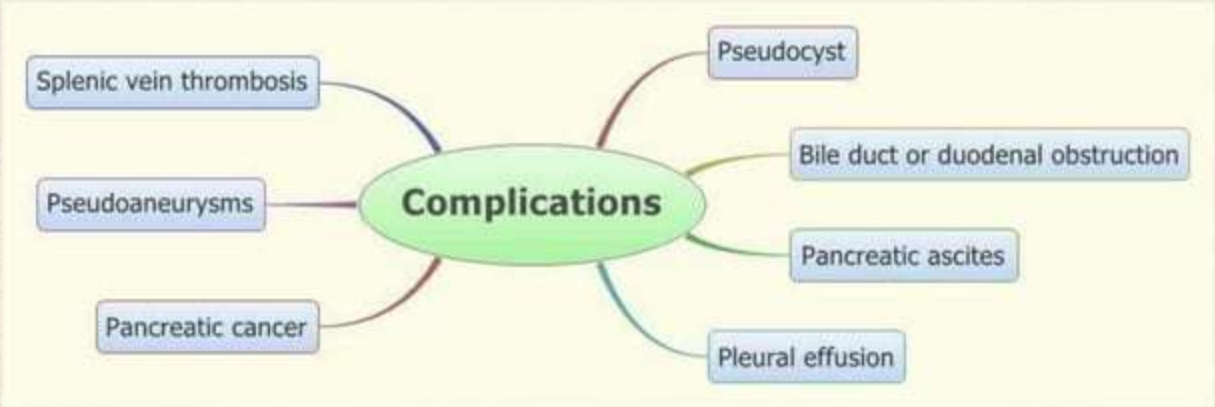
Clinical Presentation

- **Stage A** - 85%, recurrent/acute episodic pain with weight loss
- **Stage B** - Severe prolonged progressive pain with impaired pancreatic function with cholestasis, pseudocyst.
- **Stage C** - Severe exocrine / endocrine deficiency. less severe pain, complication like pseudocysts & obstruction

Complications






Complications
Splenic vein thrombosis
Pseudoaneurysms
Pancreatic cancer
Pseudocyst
Bile duct or duodenal obstruction
Pancreatic ascites
Pleural effusion


THANK YOU!!