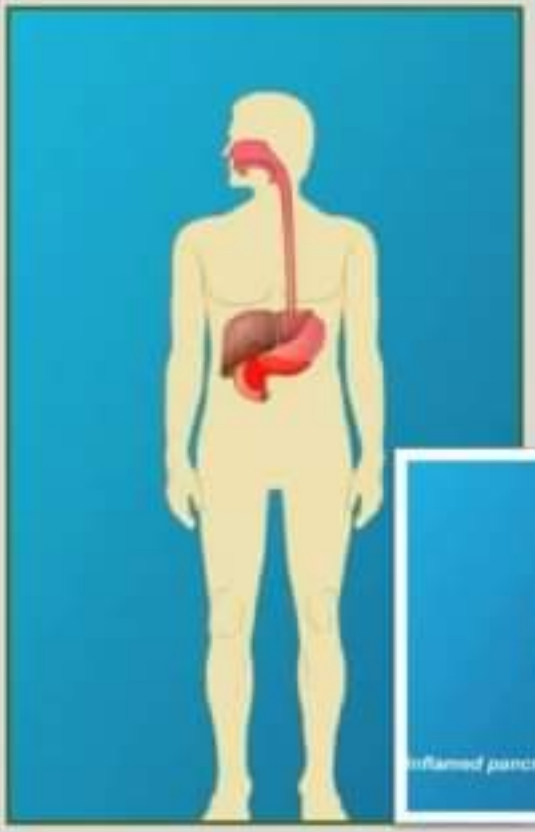


ACUTE PANCREATITIS

INTERESTING MEDICINE

Lab investigation

Amylase and lipase

- Elevated serum amylase and lipase levels, in combination with severe abdominal pain, often trigger the initial diagnosis of acute pancreatitis.
- Serum lipase rises **4 to 8 hours** from the onset of symptoms and normalizes within 7 to 14 days after treatment.
- Marked elevation of serum amylase level during first **24 hours**
- Reasons for false positive elevated serum amylase include **salivary gland disease** (elevated salivary amylase) and **macroamylasemia.**
- If the lipase level is about **2.5 to 3 times** that of Amylase, it is an indication of pancreatitis due to **Alcohol or gallstone**

Lab investigation

- Full blood count: neutrophil leucocytosis
- Electrolyte abnormalities include hypokaemia, hypocalcemia
- Elevated LDH in biliary disease
- Glycosuria (10% of cases)
- Blood sugar: hyperglycaemia in severe cases
- Ultrasound look for stones in biliary tract diseases.
- Abdominal CT scan may reveal **phlegmon**(inflammatory mass), pseudocyst or abscess(complications of acute pancreatitis)

Ranson Score

Predicting the severity of acute pancreatitis

At admission

- age in years >55 years
- white blood cell count >16000 cells/mm3
- blood glucose >11 mmol/L (>200mg/dL)
- serum AST >250 IU/L
- serum LDH >350 IU/L

At 48 hours

- Calcium (serum calcium <2.0 mmol/L (<8.0mg/dL)
- Hematocrit fall >10%
- Oxygen (hypoxemia PO2 <60 mmHg)
- BUN increased by 1.8 or more mmol/L (5 or more mg/dL) after IV fluid hydration
- Base deficit (negative base excess) >4 mEq/L
- Sequestration of fluids >6L

APACHE II score

(Acute Physiology And Chronic Health Evaluation)

Score 0 to 2: 2% mortality Score 3 to 4: 15% mortality

Score 5 to 6: 40% mortality Score 7 to 8: 100% mortality

- Hemorrhagic peritoneal fluid
- Obesity
- Indicators of organ failure
- Hypotension (SBP <90 mmHG) or tachycardia >130beat/min
- PO_2 <60 mmHg
- Oliguria (<50 mL/h) or increasing BUN and creatinine
- Serum calcium <1.90 mmol/L (<8.0mg/dL)
- serum albumin <33 g/L (<3.2.g/dL)>

Balthazar scoring

Balthazar Grade

Balthazar Grade	Appearance on CT	CT Grade Points
Grade A	Normal CT	**0 points**
Grade B	Focal or diffuse enlargement of the pancreas	**1 point**
Grade C	Pancreatic gland abnormalities and peripancreatic inflammation	**2 points**
Grade D	Fluid collection in a single location	**3 points**
Grade E	Two or more fluid collections and / or gas bubbles in or adjacent to pancreas	**4 points**

Necrosis Score

Necrosis Percentage	Points
No necrosis	**0 points**
0 to 30% necrosis	**2 points**
30 to 50% necrosis	**4 points**
Over 50% necrosis	**6 points**

The numerical CTSI (Computed Tomography Severity Index) has a maximum of ten points, it is the sum of the Balthazar grade points and pancreatic necrosis grade points.

Management

- Iv fluid replacement(normal saline)
- Bowel rest (NG tube, NPO) in severe case
- Administration of meperidine/pethidine as pain killer.
- Antiemetic if necessary
- Monitor & correct electrolytes.
- Prevent infection by antibiotic prophylaxis.
- Determine & treat specific etiology(avoid alcohol)
- Indication to surgery if pancreatitis not respond to treatment.

Complications

- **Immediate**
  - **Shock**
  - **Acute respiratory distress syndrome** :Pulmonary failure in acute pancreatitis is believed to be caused by circulating activated digestion enzymes (e.g. trypsin, phospholipase A2, etc.) leading to a loss of surfactant, atelectasis and irritation eventually leading to ARDS and pleural effusion.

- **Late**
  - Pancreatic pseudocyst
  - Pancreatic abscess
  - Pancreatic necrosis
  - Progressive jaundice
  - Persistent duodenal ileus
  - GI bleeding
  - Pancreatic ascites

Recommendations

AVOID

Alcohol and tobacco

High-fat foods and triglycerides

Medication

DO

Stay hydrated

Maintain healthy diet

Eat fruits and vegetables

Definition

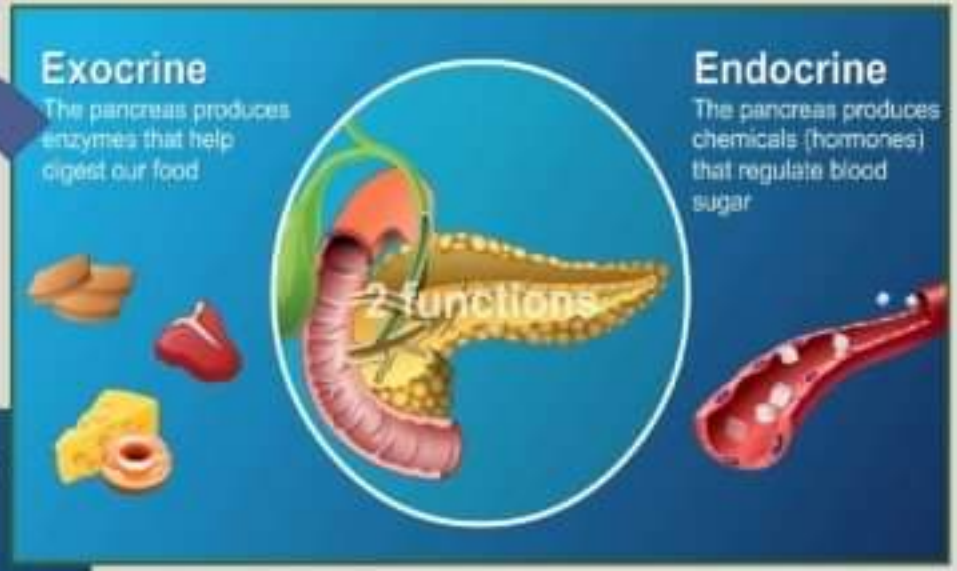
- Acute pancreatitis (AP), defined as the acute nonbacterial inflammatory condition of the pancreas, is derived from the early activation of digestive enzymes found inside the acinar cells, with variable compromise of the gland itself, nearby tissues and other organs.

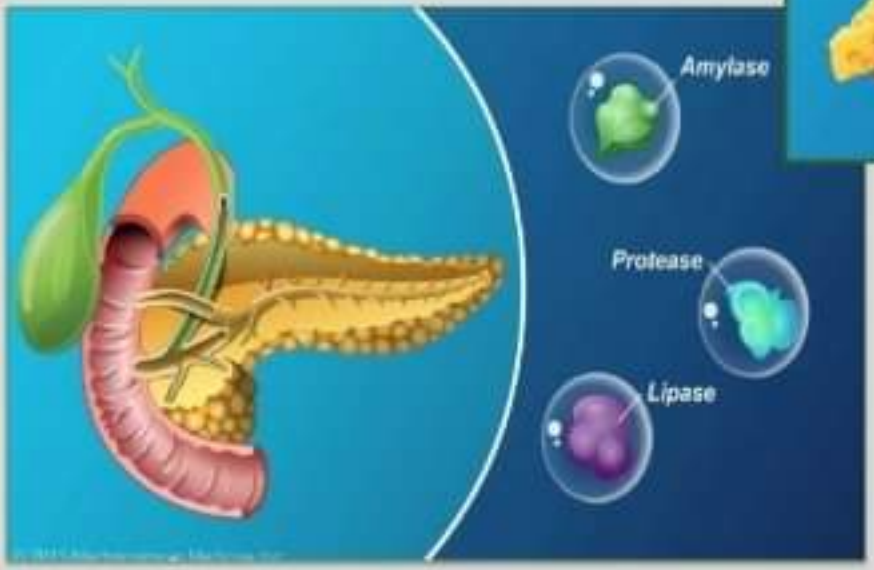
Incidence

- Male:female ratio is 1:3- in those with gallstones and 6:1 in those with alcoholism

-

Functions





Exocrine

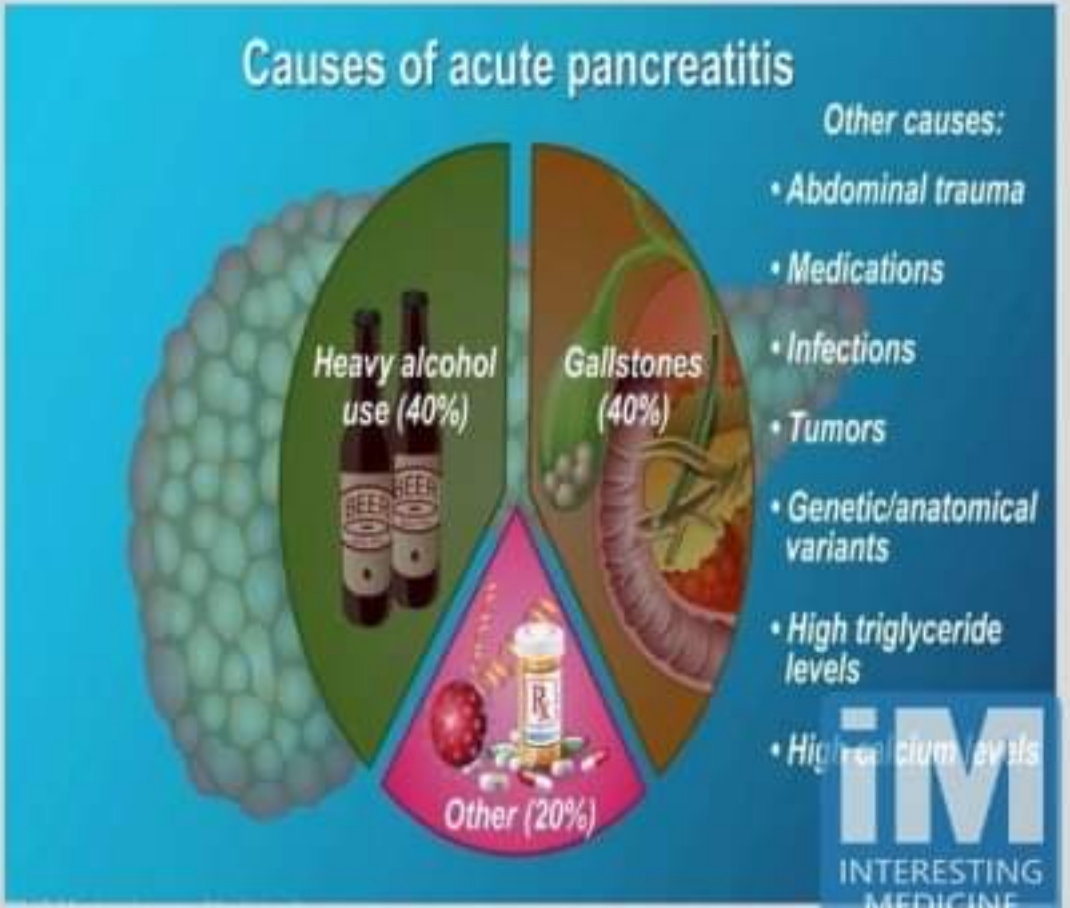
Amylase
Protease
Lipase

Endocrine

Insulin
Somatostatin
Glucagon

Causes

- **Non-traumatic(75%)**
  - **Biliary tract diseases**
  - **Alcohol**
  - Viral infection(EBV, CMV, mumps)
  - Drugs(steroid, thiazide, furosemide)
  - Scorpion bites
  - Hyperlipidemia
  - Hyperparathyroidism
- **Traumatic (5%)**
  - Operative trauma
  - Blunt/penetrating trauma
  - Lab test(ERCP / angiography)
- **Idiopathic(20%)**



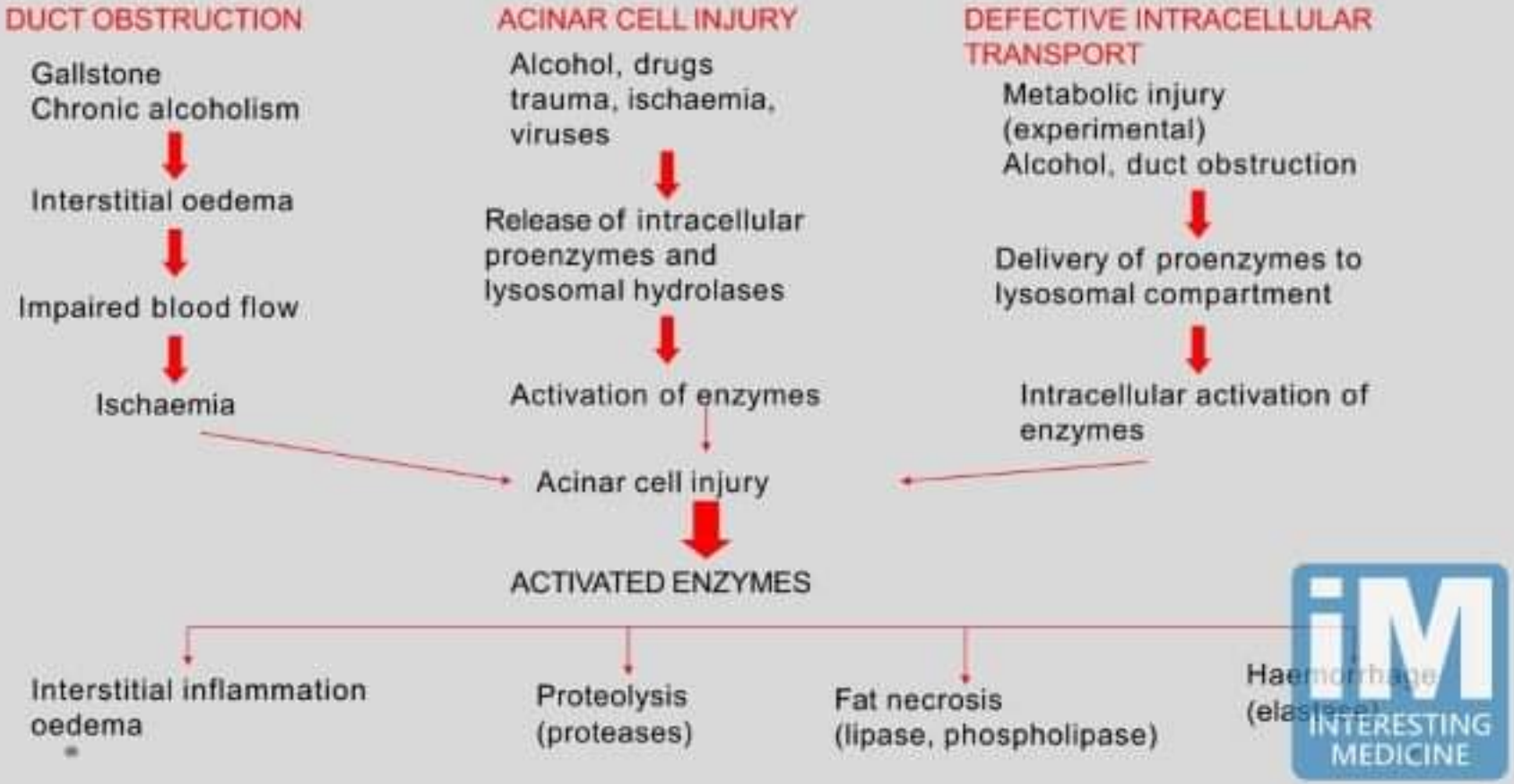
Symptoms and signs

- The most common symptoms and signs include:
  - Severe **epigastric pain** radiating to the back, relieved by leaning forward
  - **Nausea, vomiting, diarrhea and loss of appetite**
  - **Fever/chills**
  - Hemodynamic instability, including **shock**
  - In severe case may present with **tenderness, guarding, rebound.**

Signs which are less common, and indicate severe disease, include:

- Grey-Turner's sign (hemorrhagic discoloration of the flanks)
- Cullen's sign (hemorrhagic discoloration of the umbilicus)

Pathogenesis of acute pancreatitis



Pathologic Activity of activated Enzymes

Proteases : digest the walls of blood vessel → blood extravasation;

Amylase :is released into the blood (but is a nonspecific diagnostic marker).

Released lipases : (better diagnostic marker) cause fat necrosis within abdomen and subcutaneous tissue, can → discolouration of skin (Grey Turner's sign).

Released fatty acids :can bind Ca^{2+} can →hypocalcaemia.

Destruction of adjacent islets can → hyperglycaemia and thus cause Type II diabetes.

Normal pancreas

Acute pancreatitis

Acute Pancreatitis; Haemorrhage and necrosis