

# Acute pancreatitis

# Definition

- “Acute pancreatitis”:
- A reversible inflammation of the pancreas as a result of auto digestion by its own enzymes. It appears suddenly (an emergency) and lasts for days.
- OR: An acute condition presenting with abdominal pain, a threefold or greater rise in the serum levels of the pancreatic enzymes ((amylase or lipase)), and/ or characteristic findings of pancreatic inflammation on contrast-enhanced CT.

# Epidemiology

- Accounts for 3% of all cases of abdominal pain among patients admitted to hospital in the UK.
- The disease may occur at any age, with a peak in young men and older women.

# Epidemiology

- The incidence of acute pancreatitis varies between populations. In large population studies from Scotland and Finland the incidence of the disease has risen steadily to the current 400 patients/million per year.
- (Biliary + alcoholic) 90%
- Even in the west, biliary pancreatitis is the most prevalent type.

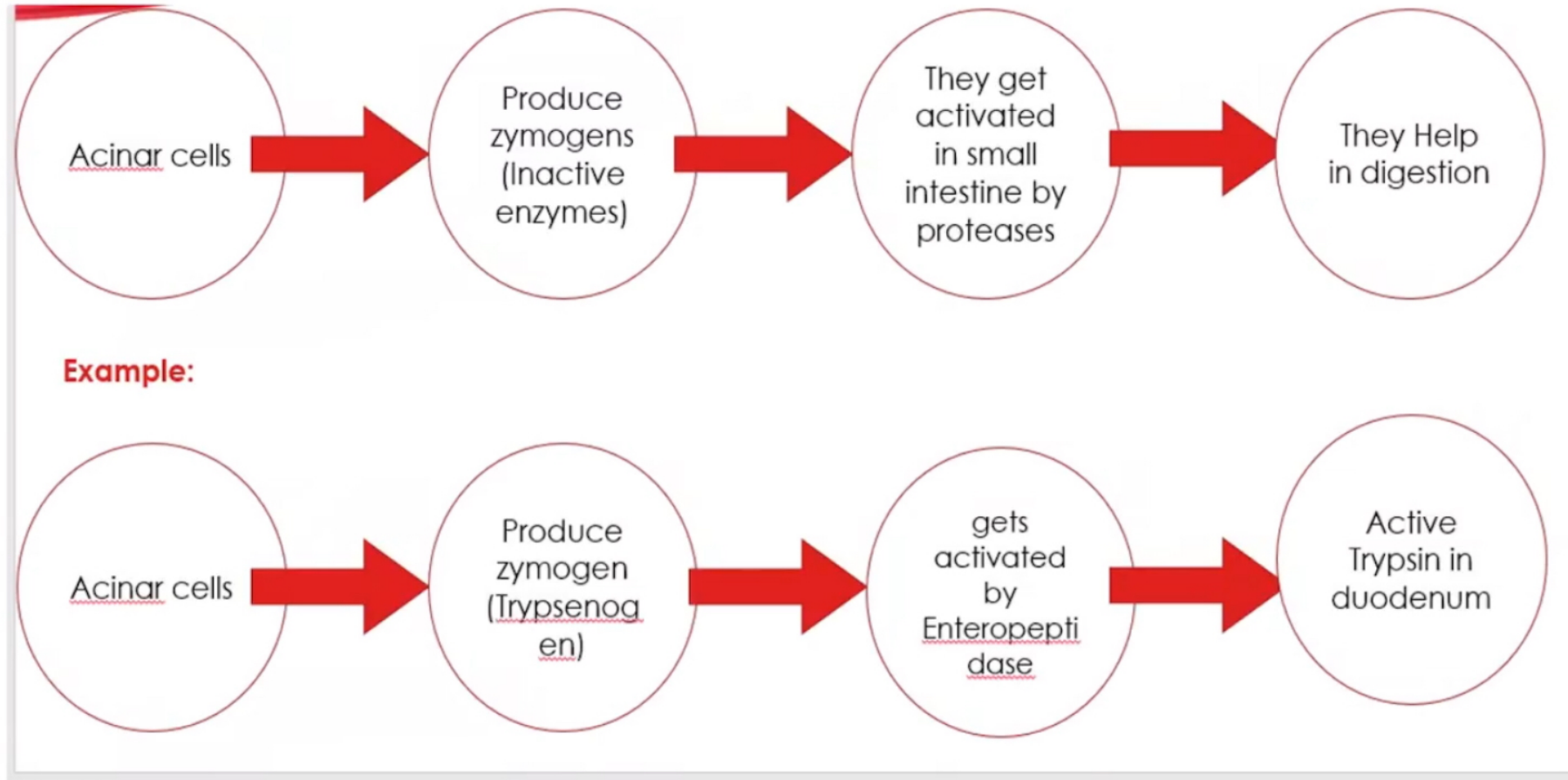
# Epidemiology

- Overall mortality is from 2.0 to 7.5%, highest in those who are over 70 years, obese individuals, and those with comorbidity at the time of onset.
- Prospective and retrospective studies record 45 to 50% of deaths as occurring in the initial week of the illness secondary to fulminant multiple organ failure

# Causes

- Idiopathic
- **Gallstones**
- **Ethanol (alcohol)**
- Trauma (usually a penetrating one)
- Steroids
- Mumps(viral)
- Autoimmune disease (ex: polyarteritis nodosa).
- Scorpion bite (Rare)
- Hyperlipidemia/ Hypercalcemia
- ERCP [endoscopic retrograde cholangio-pancreatography] → iatrogenic
- Drugs (diuretics, Isonazid {INH}, reverse transcriptase inhibitors and Metronidazole).

# PATHOPHYSIOLOGY



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- Any early activation of the pancreatic pro-enzymes ( zymogens) leads to acute pancreatitis due to auto-digestion



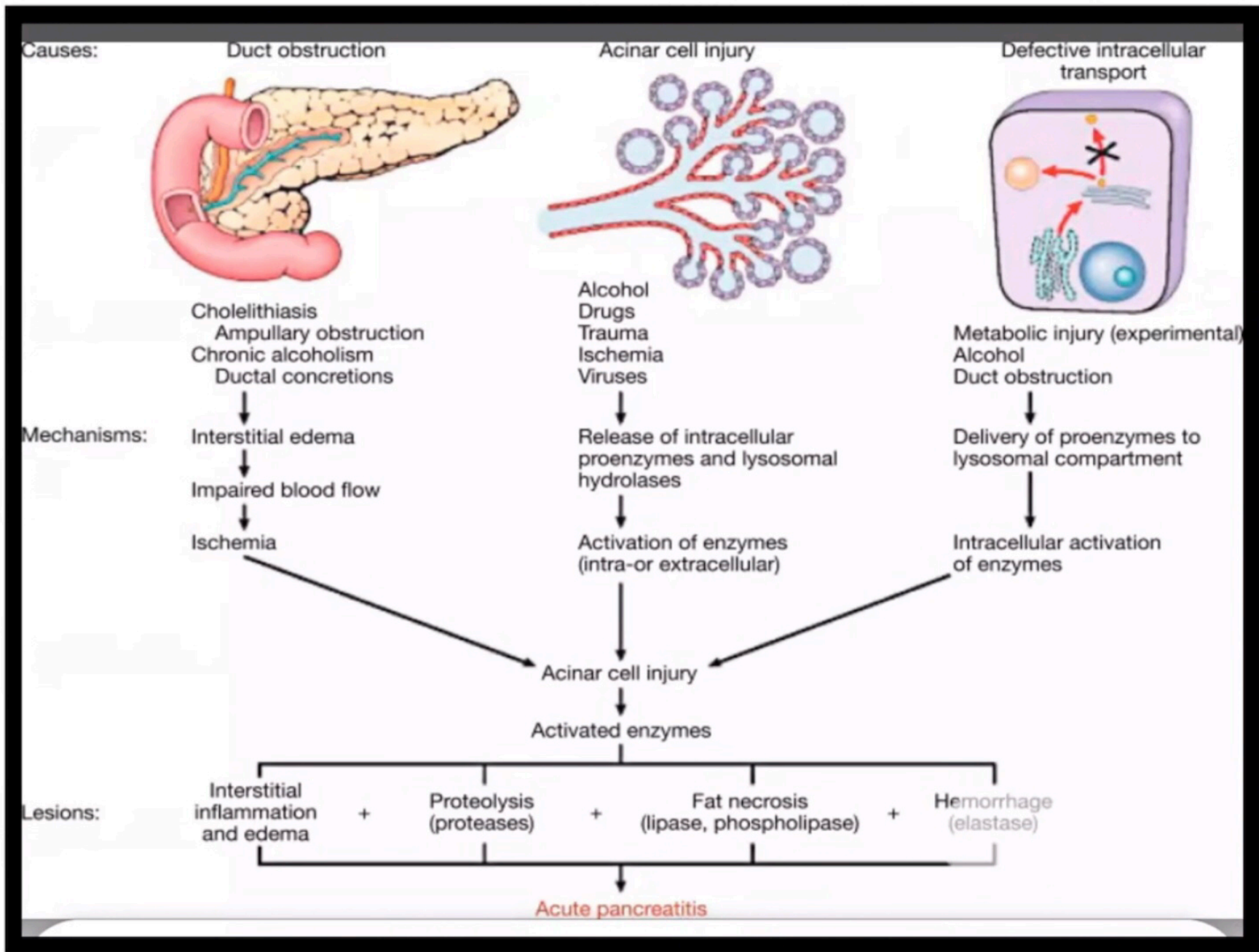
# PATHOPHYSIOLOGY

- An initial cause
- Injury to the pancreatic ductal cells
- Cell membrane trafficking problem (signaling error)
- Early activation of pancreatic enzymes
- Destruction of the pancreatic tissue.
- Cell injury causes the release of activated neutrophils which produces proteolytic enzymes and activation of zymogens.

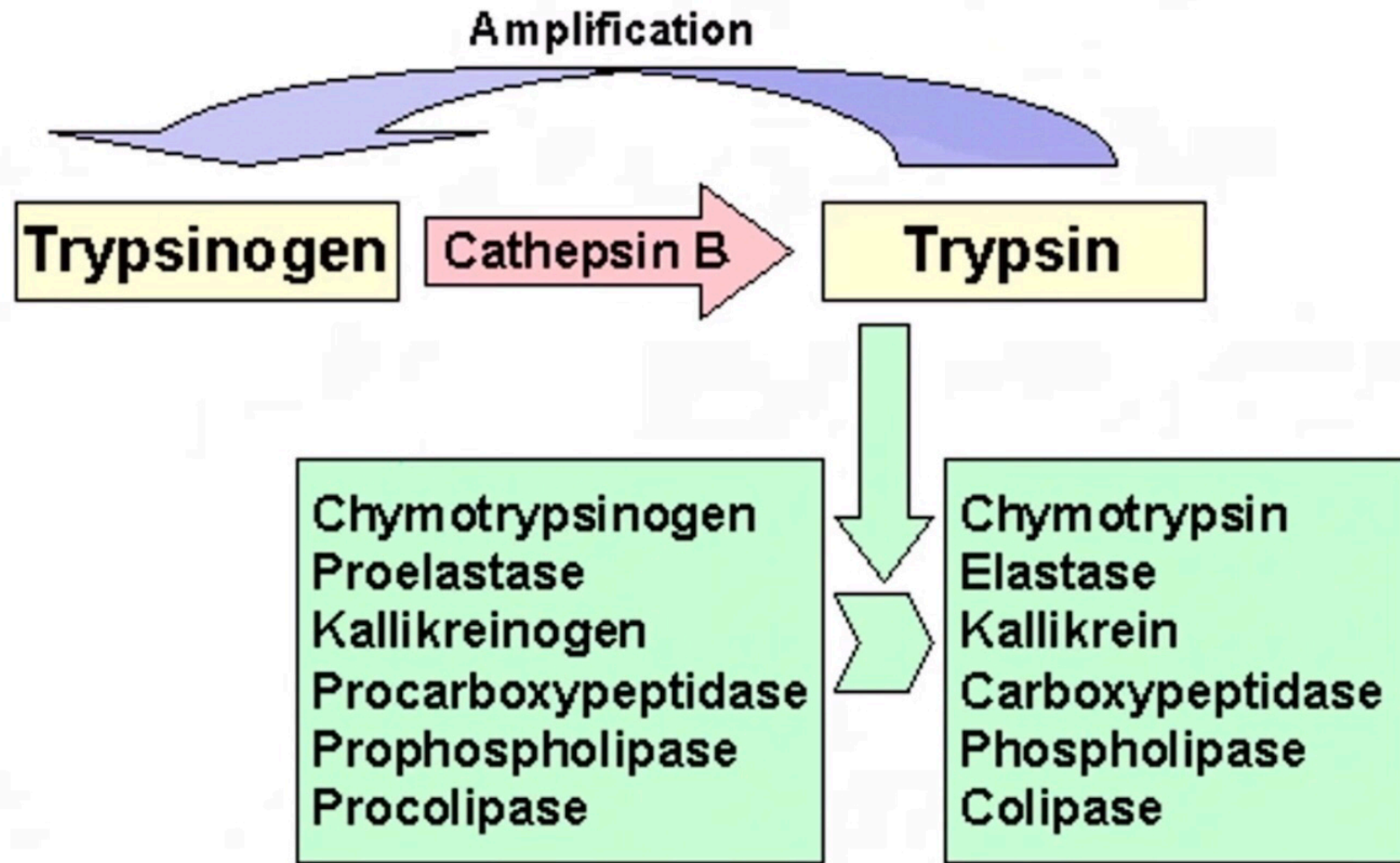
# PATHOPHYSIOLOGY

Cells injury may take place as a result of any cause  
Once cellular injury has been initiated, the inflammatory process can lead to pancreatic edema, haemorrhage and, eventually, necrosis.

As inflammatory mediators are released into the circulation, systemic complications can arise such as haemodynamic instability, acute respiratory distress syndrome and pleural effusions.



# PATHOPHYSIOLOGY



# Presentation

- The cardinal symptom of acute pancreatitis is **abdominal pain**
- **dull, boring, and steady**
- **sudden in onset** generally following substantial meal and gradually **intensifies in severity until reaching a constant ache.**
- located in the **upper abdomen**, usually in the epigastric region
- The pain **radiates directly** through the abdomen **to the back** in approximately one half of cases
- Nausea and vomiting

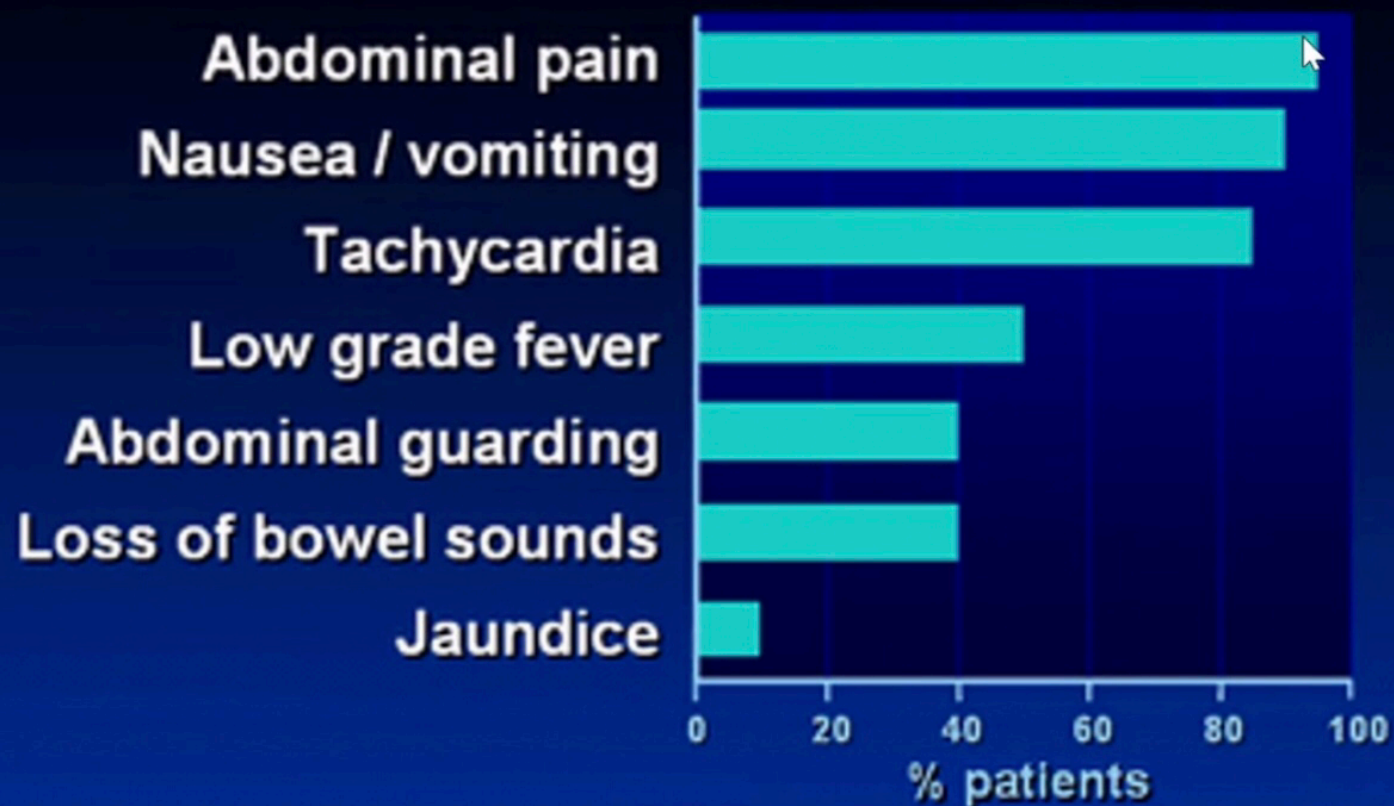
# Presentation

- Ask the patient about recent operative or other invasive procedures (eg, endoscopic retrograde cholangiopancreatography [**ERCP**] or **family history of hypertriglyceridemia**). Patients frequently have a **history of previous biliary colic and binge alcohol consumption**, the major causes of acute pancreatitis.

- Appearance: Gravely ill with profound shock, toxicity and confusion in severe cases
- Fever (76%) and tachycardia (65%) are common abnormal vital signs; hypotension may be noted

## Acute Pancreatitis

### Presenting Features





# Diagnosis

- The diagnosis of AP is most often established by the presence of 2 of the 3 following criteria: (i) abdominal pain consistent with the disease, (ii) serum amylase and / or lipase greater than three times the upper limit of normal, and / or (iii) characteristic findings from abdominal imaging

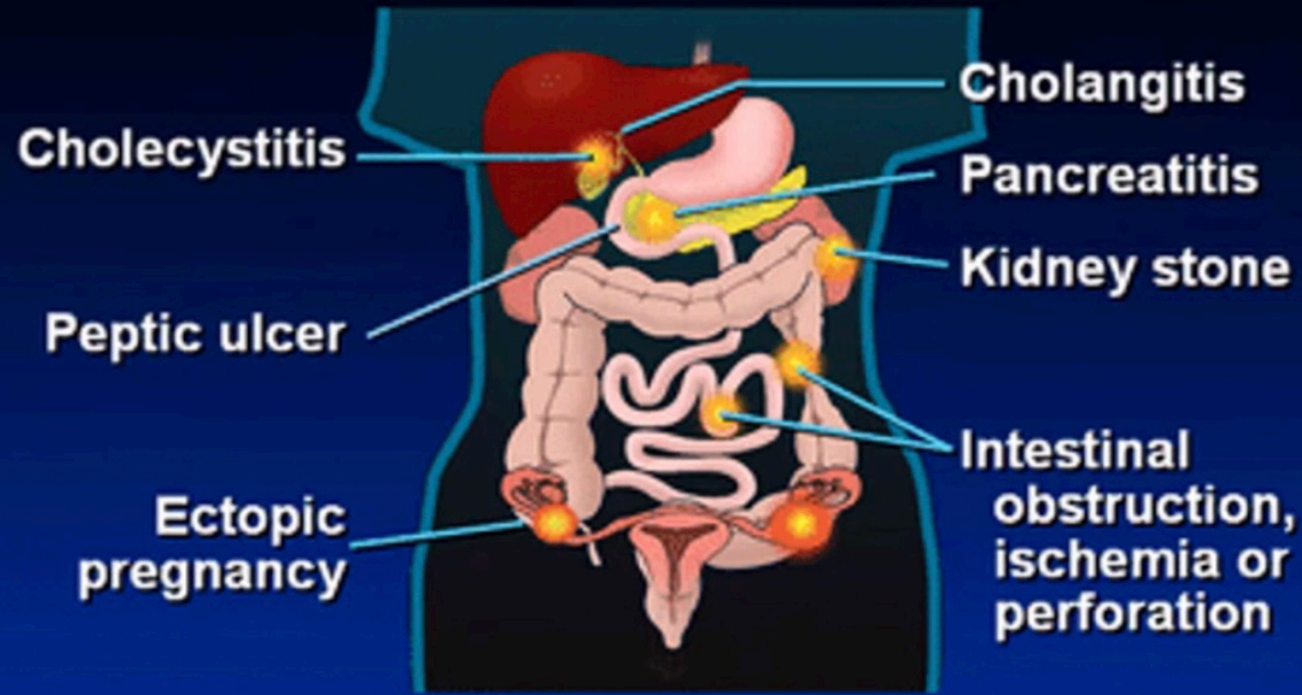
# Diagnosis

- Serum markers
  - Amylase
    - Easiest to measure and most widely used
    - Rises immediately
    - Peaks in few hours
    - Remains for 3-5 days
    - “Three fold rise is diagnostic”
    - May be normal in severe attacks
    - May be falsely negative in hyperlipidemic patients
    - Inverse correlation between severity and serum amylase level
    - No need to repeat

# Causes of hyperamylesemia

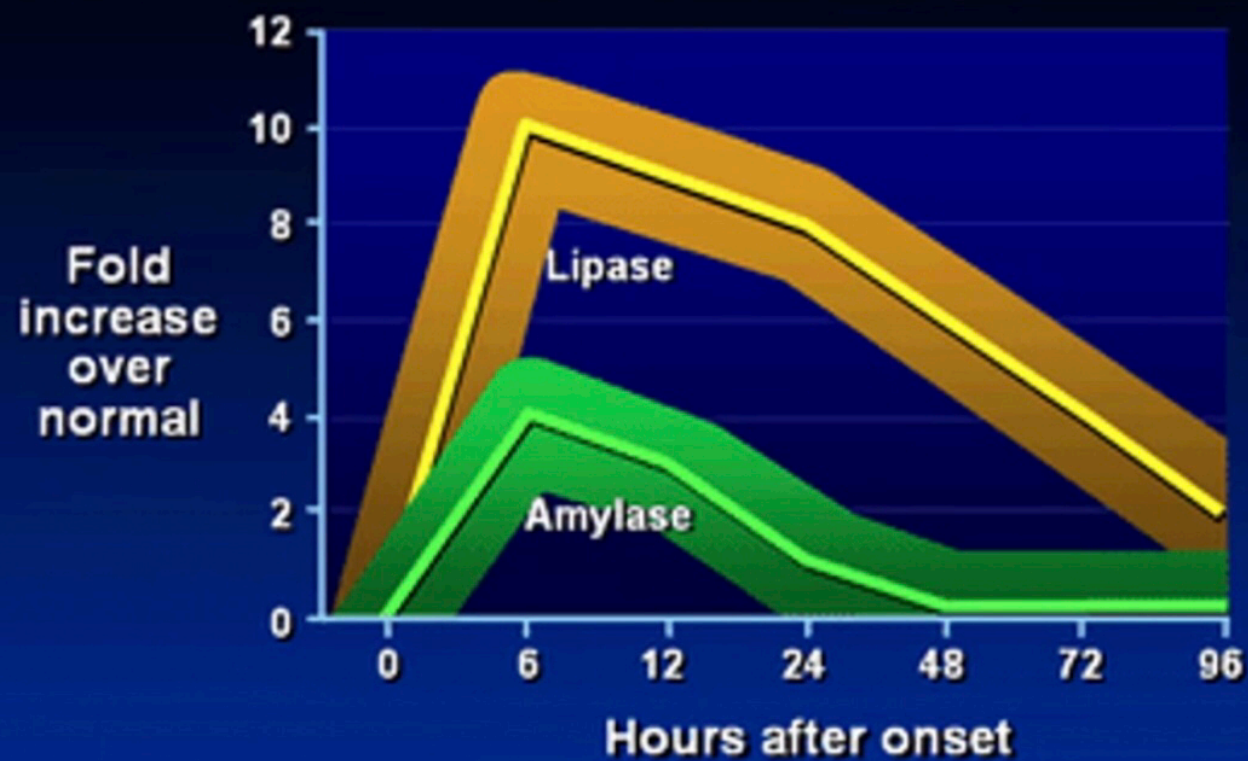
**Acute Pancreatitis**

## **Elevated Serum Amylase**



## Acute Pancreatitis

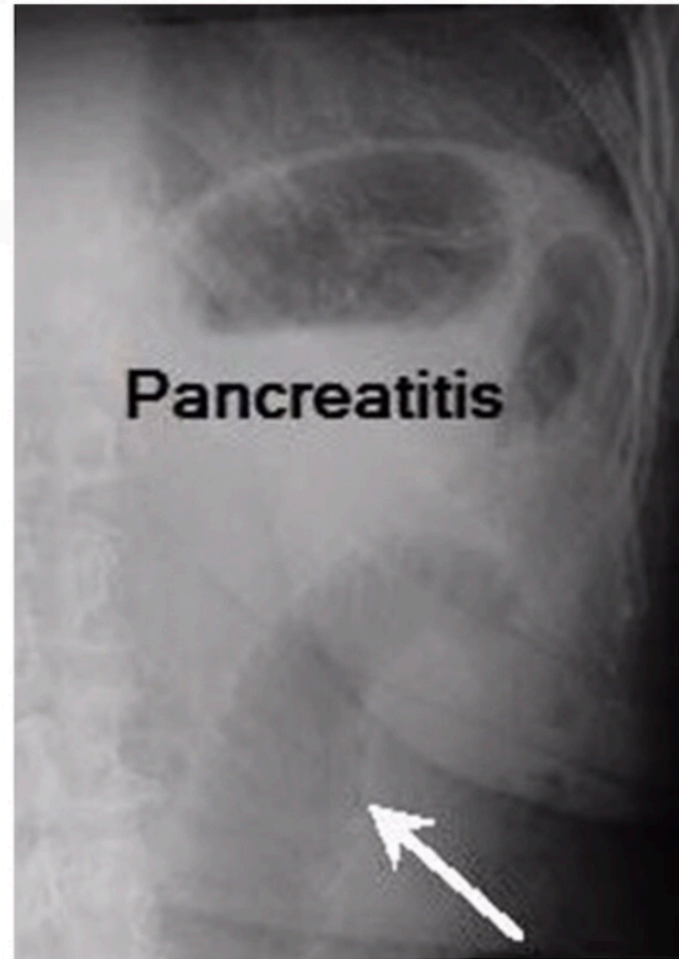
### Time Course of Enzyme Elevations



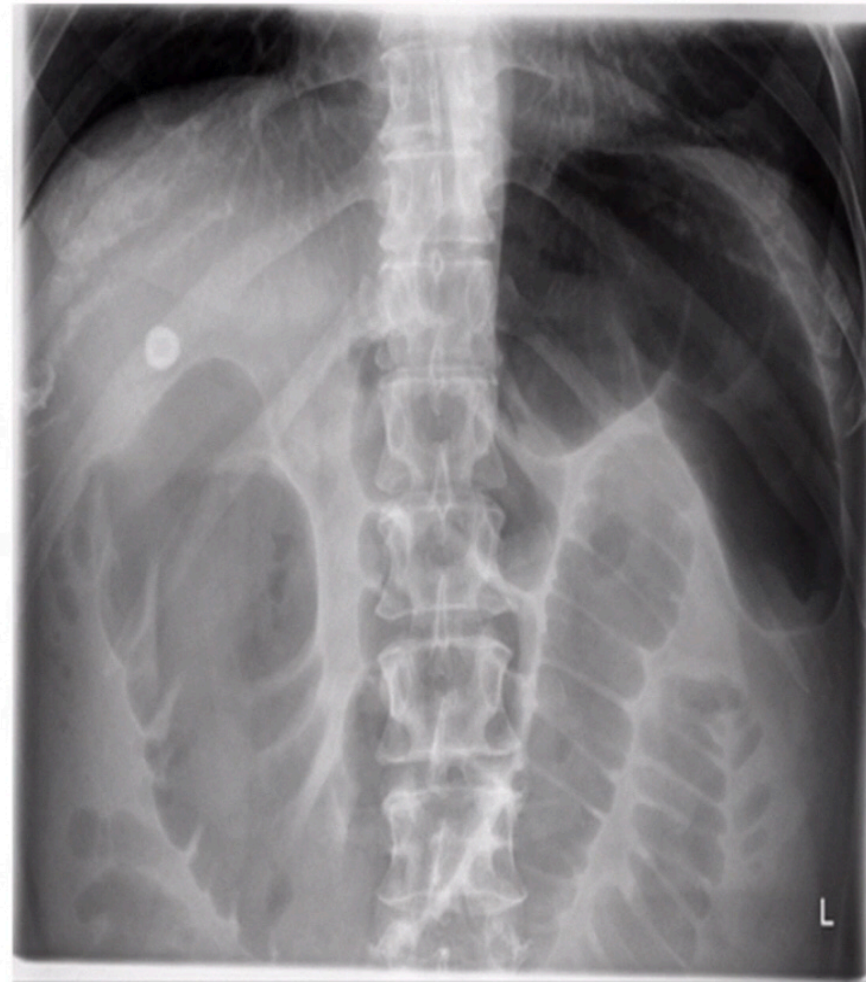
# Radiology

- X-ray
  - Air in the duodenal C loop
  - Sentinel loop sign
  - Colon cutoff sign
  - All these signs are **non specific**

## Sentinel loop

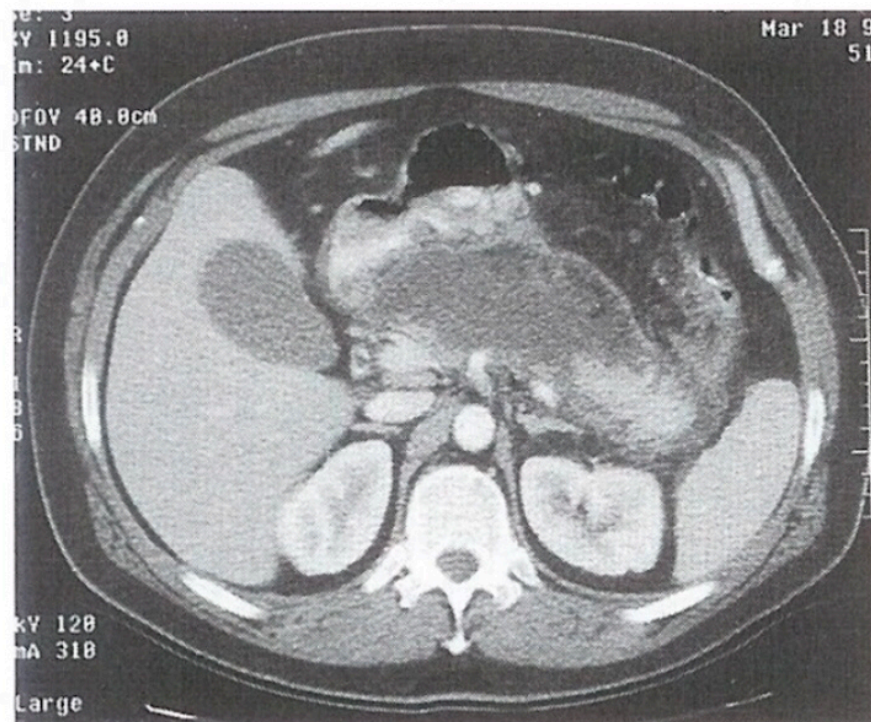


## Colon cutoff sign



# Radiology

- CE-CT
  - Enlargement of the pancreas
    - (focal/diffuse)
  - Irregular enhancement
  - Shaggy Pancreatic contour
  - Thickening of fascial planes
  - fluid collections.
    - Intraperitoneal / retroperitoneal



# Radiology

- U/S
  - Diagnosis of gallstones
  - F/U of pseudocysts



# PROGNOSIS

- Course either mild or severe
  - Mild = edematous pancreatitis
  - Severe = necrotic pancreatitis or hemorrhagic pancreatitis
- Most episodes of AP are mild and self-limiting, needing only brief hospitalization.
- In patients with severe disease, two phases of AP are recognized: early (within the first week) and late.

# PROGNOSIS

- Serum markers
- CT
- Systemic complications
- Prognostic scores
  - Ranson
  - Apache II
  - Modified Glasgow
  - Atlanta

## Atlanta Revision (2013)

### Mild acute pancreatitis

Absence of organ failure

Absence of local complications

### Moderately severe acute pancreatitis

1. Local complications **AND/OR**
2. Transient organ failure (<48h)

### Severe acute pancreatitis

Persistent organ failure >48h<sup>a</sup>

GI bleeding (>500cc/24 hr)

Shock – SBP  $\leq$  90 mm Hg

PaO<sub>2</sub>  $\leq$  60%

Creatinine  $\geq$  2 mg/dl

## Predicting severe AP

### Clinical findings associated with a severe course for initial risk assessment a

#### *Patient characteristics*

Age >55 years (53,57)

Obesity (BMI >30 kg/m<sup>2</sup>) (68)

Altered mental status (69)

Comorbid disease (53)

#### *The systemic inflammatory response syndrome (SIRS) (6,53,54,70,71)*

Presence of >2 of the following criteria:

- pulse >90 beats/min

- respirations >20/min or PaCO<sub>2</sub> >32 mm Hg

- temperature >38°C or <36°C

-WBC count >12,000 or <4,000 cells/mm<sup>3</sup> or >10% immature neutrophils (bands)

#### *Laboratory findings*

BUN >20 mg/dl (63)

Rising BUN (63)

HCT >44% (62)

Rising HCT (62)

Elevated creatinine (72)

#### *Radiology findings*

Pleural effusions (73)

Pulmonary infiltrates (53)

Multiple or extensive extrapancreatic collections (67)

# PROGNOSIS (Scoring systems)

**Table 32-4**

## **Ranson's Prognostic Signs of Pancreatitis**

### **Criteria for acute pancreatitis not due to gallstones**

#### *At admission*

Age >55 y

WBC >16,000/mm<sup>3</sup>

Blood glucose >200 mg/dL

Serum LDH >350 IU/L

Serum AST >250 U/dL

#### *During the initial 48 h*

Hematocrit fall >10 points

BUN elevation >5 mg/dL

Serum calcium <8 mg/dL

Arterial PO<sub>2</sub> <60 mm Hg

Base deficit >4 mEq/L

Estimated fluid sequestration >6 L

### **Criteria for acute gallstone pancreatitis**

#### *At admission*

Age >70 y

WBC >18,000/mm<sup>3</sup>

Blood glucose >220 mg/dL

Serum LDH >400 IU/L

Serum AST >250 U/dL

#### *During the initial 48 h*

Hematocrit fall >10 points

BUN elevation >2 mg/dL

Serum calcium <8 mg/dL

Base deficit >5 mEq/L

Estimated fluid sequestration >4 L

AST = aspartate transaminase; BUN = blood urea nitrogen; LDH = lactate dehydrogenase; PO<sub>2</sub> = partial pressure of oxygen; WBC = white blood cell count.

SOURCE: Reproduced with permission from Ranson JHC: Etiological and prognostic factors in human acute pancreatitis: A review. *Am J Gastroenterol* 77:633, 1982.

# PROGNOSIS (CT)

- “CT scanning with bolus IV contrast has become the gold standard for detecting and assessing the severity of pancreatitis”
- “Currently, IV bolus contrast enhanced CT scanning is routinely performed on patients who are suspected of harboring severe pancreatitis, regardless of their Ranson’s or APACHE scores”

# PROGNOSIS (CT)

- Balthazar CT-severity index (CTSI)
  - CTSI considers degree of necrosis
  - Also considers the CT grade
  - A final score is given and correlates with mortality and complication development

# PROGNOSIS (CT)

- Balthazar grading
  - Grade A - Normal-appearing pancreas 0
  - Grade B - Enlargement of the pancreas 1
  - Grade C - Pancreatic gland abnormalities with peripancreatic fat infiltration 2
  - Grade D - A single fluid collection 3
  - Grade E - Two or more fluid collections 4



# PROGNOSIS (CT)

- Grade of necrosis and the points assigned per grade are as follows:

– None	0 points
– Grade 0.33	2 points
– Grade 0.5	4 points
– Grade > 0.5	6 points

# PROGNOSIS (CT)

CTSI	Mortality	Complications
0-3	3%	8%
4-6	6%	35%
7-10	17%	92%

# PROGNOSIS

- Mild is defined as:
  - No systemic complications
  - Low APACHE/Ranson scores
  - CE-CT findings (Balthazar)
  - CRP level <150

# MANAGEMENT

- Core of treatment based on
  - Physiological monitoring
  - Metabolic support
  - Maintenance of fluids and electrolytes

# MANAGEMENT (Mild)

- Mainstay of management is supportive
  - NPO
  - IVF
- When to resume oral intake?
  - Absence of pain
  - Absence of tenderness
  - Patient feeling hungry
- On average takes about 3-7 days
- Sips of water and build up to low protein low fat diet

# MANAGEMENT (Mild)

- **Proved of no benefit**
- N/G tube
- H2 blockers
- Anti-secretory agents ( eg. Somatostatin )
- Antibiotic therapy in the absence of signs or documented sources of infection

# Management (severe)

- **Sterile Necrosis**

- No sys. Comp., no infec. (i.e. uncomplicated)
  - Supportive + Prophylactic Abx
- Sys. Comp. + infection? ( mild complication)
  - CT guided aspiration → gram stain/culture → Abx
- Mult. Sys comp + toxicity/shock (frank complication)
  - surgical debridement

# Management (severe)

- Nutritional support
  - NPO with resumption of diet when fit
  - If NPO > 7 days...
  - TPN vs. Jujenal tube feeding?
    - TPN: gastric mucosal atrophy → bacterial translocation
    - Jujenal tube feeding: induces pancreatic secretion
  - Inconclusive studies:
    - Jujenal T. feeding is superior



# Biliary Pancreatitis

- **Bilirubin Dropping**
- Lap. Cholecystectomy and I/O cholangiogram ( same admission)
- **Bilirubin Persists**
- MRCP to confirm presense of stone thin ERCP
- Lap. Cholecystectomy

# **Pancreatic Pseudocyst**

- **Pseudocysts are encapsulated localized collection of pancreatic enzyme, inflammatory fluid and necrotic debris on pancreas or in part or the whole of the lesser sac.**
- **They are distinguished from other types of pancreatic cysts by their lack of an epithelial lining.**

# **Pancreatic Pseudocyst**

- **CT scan is the investigation of choice in pancreatic pseudocysts. It has a sensitivity of 90-100%**
- **All cysts do not require treatment. In many cases the pseudocysts may improve and go away on their own.**
- **In a patient with a small (less than 5cm) cyst that is not causing any symptoms, careful observation of the cyst with periodic CT scans is indicated.**

# **Pancreatic Pseudocyst**

- **Percutaneous catheter drainage**
- **Internal Drainage**

**Surgical**

**Endoscopic**