



# Biochemical Markers in Acute Abdomen

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# Acute Abdomen

- Definition

Patient is acutely ill with signs and symptoms related to abdomen

- 20-40% admission rates

- 50-65% inaccurate initial diagnosis

De Dombal FT. Diagnosis of acute abdominal pain. New York: Churchill Livingstone; 1991.

# MAGNITUDE OF THE PROBLEM

- **70-80% - Edematous Interstitial Inflammation (Mild and Self limiting)**
- **20-25% Necrotising Pancreatitis**
  - **20-30% will develop local or systemic complications**
- **Approx 1 in 4 pts who develop complications will die**

# Acute Abdomen

> 1000 causes exist

- **Non Specific Abdominal Pain (34%)**
- Acute appendicitis (28%)
- **Acute cholecystitis (10%)**
- Subacute Bowel Obstruction (4%)
- Perforated Peptic Ulcer (3%)
- **Pancreatitis (3%)**
- Diverticular disease (2%)
- Others (16%)

De Dombal FT, Margulies M. Acute abdominal pain. Surgery 1996;

# Biochemical markers

“SURGERY IS ALWAYS SECOND BEST ; IF YOU CAN DO SOMETHING ELSE, IT’S BETTER”

- John Kerklin

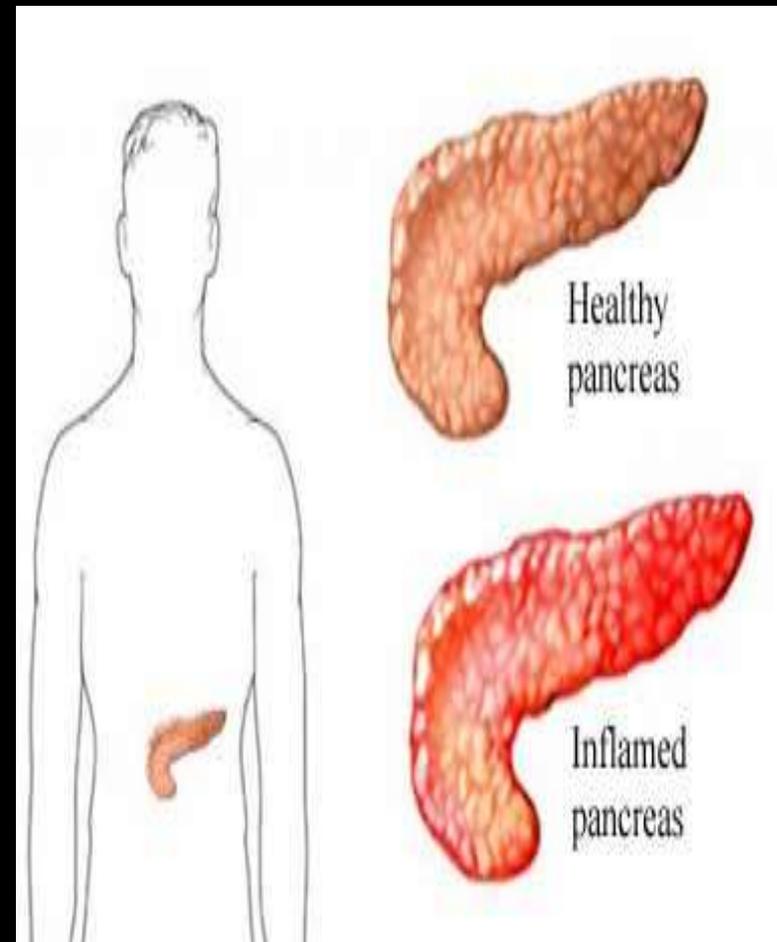
- What is the correct diagnosis?
- What is the prognosis?
- Are complications developing?
- Can an associated condition identified?
- What is the ideal timing for surgery?

Rapid and quick decisions – rarely necessary and if carried out may be incorrect or misleading

Delay leads to fatal outcome

# What is pancreatitis?

- Acute inflammation of the pancreas
- Varying degree of regional tissue involvement and remote organ systems
- Unless there is evidence of chronic pancreatitis, considered as exacerbation of inflammation superimposed on chronic pancreatitis



# PANCREATITIS

- MILD uncomplicated recovery

- SEVERE (Acute Pancreatitis) with evidence of failure of one or more systems or local complication.



These terms are defined retrospectively, when outcome is known

Prospectively defined on the basis of scoring systems. Predicted Mild or Predicted Severe

# Causes of Acute Pancreatitis

➤ **Gall stone (45 %)**

➤ **Ethanol abuse (Alcohol )  
(35 %)**

➤ **Idiopathic ( 10 % )  
Autoimmune**

● **Other ( 10 % ) => include**

## Trauma

Postendoscopic retrograde  
cholangiopancreatography

Pancreatic malignancy , PUD ,IBD

## Medications

Thiazide Tetracycline Sulphonamid  
Corticosteroid etc

## Metabolic

- Hypertriglyceridemia  
,Hypercalcemia

## Infectious

-Viral , Bacterial , Parasitic

# DIAGNOSIS

**History and physical examination** – Cornerstone

**Laboratory data**

**Radiological studies**

Plain Films

**Ultrasonography**

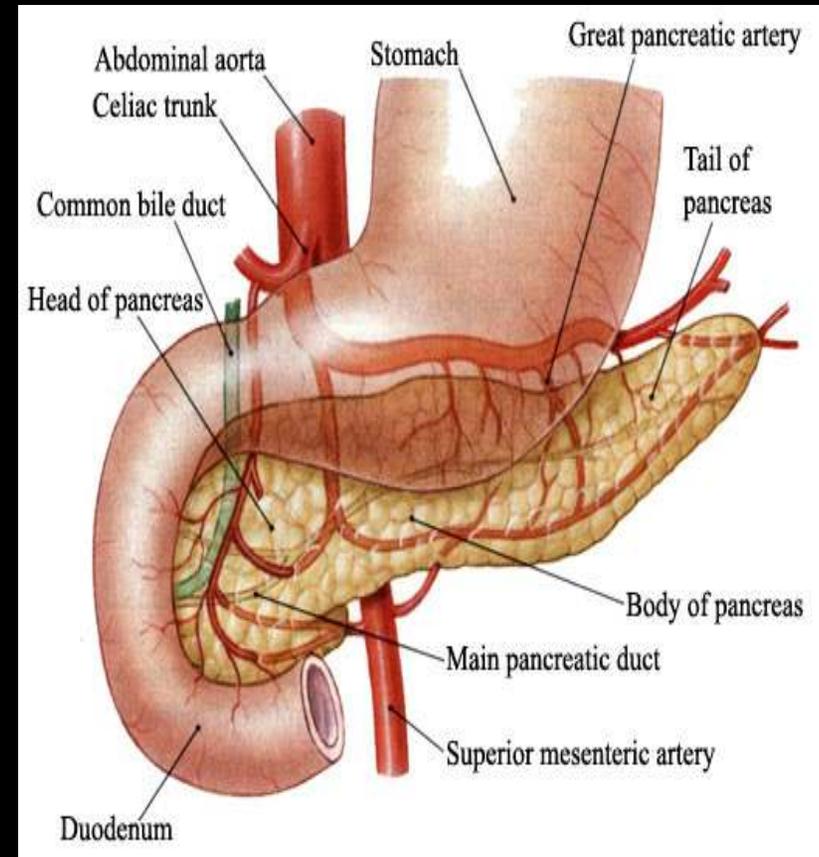
Sens: 62-95%, Specif: >95%,  
pancreas not visualized in > 40%pts

**CT scan**

Sens 90% Specif 100%

**ERCP**

**(replaced secretin stimulation test)**



# MANIFESTATIONS OF ACUTE PANCREATITIS

- LOCAL

Gnawing pain  
Pancreatitis  
Pancreatic Ca

Necrosis, infection,  
Fluid collection, abscess  
Edema, inflammation,



Cullen's sign



# NECROTISING PANCREATITIS

- o Interstitial edematous inflammation
- o Extensive necrosis of pancreatic **exocrine** and **endocrine** parenchyma,
- o Fatty necrosis of **peripancreatic** and **retroperitoneal** tissue compartment

# Systemic Inflammatory Response syndrome

**Multiorgan dysfunction.....failure.....death**

**Peripancreatic fluid collection of**

- endotoxin
- prostacyclin
- activated trypsin
- complement
- thromboxane
- phospholipase
- elastase
- IL-6,8
- Others vasoactive and toxic substances

# Local Complications

- Pancreatic necrosis;

Patchy or diffuse superficial or parenchymal necrosis, unequivocally demonstrated by inspection after opening of the pancreatic capsule , or histological criteria; local or diffuse areas of non enhancement on CT, sterile necrosis

- Infected Pancreatic necrosis;

Necrosis with positive bacterial cultures

- Pancreatic abscess;

Loculated walled off collections of pus as a late complication of AP, usually after 3 weeks

- Extension into ;

Retoperitoneum, perirenal spaces, mesocolon, major and minor omentum, mediastinum.

# Systemic Complications

- **Pulmonary**
  - Atelactasis
  - Pleural effusions
  - ARDS
- **Cardiovascular**
  - Cardiogenic shock
- **Neurologic**
  - Pancreatic encephalopathy
- **Metabolic**
  - Metabolic acidosis
  - Hypocalcemia
  - Altered glucose metabolism
- **Hematologic**
  - GI bleeding
- **Renal**
  - Prerenal failure

# Bacterial /Fungal inoculation

- Risk of bacterial infection on necrotic tissue
  - 60% in proven cases of NP
  - Risk in 1st week =25%
  - Risk in 2<sup>nd</sup> week = 35-40%
  - Risk in 3<sup>rd</sup> week =60%
- Organisms are Gram negative  
E-coli, Proteus, Pseudomonas,

# *Marker of pancreatitis injury*

## ➤ Serum amylase

- Most common
- Levels Rise within 2-12hrs,
- 3 times normal is cut off (35-118 IU/liter )
- amylase levels and sensitivity decrease with time from onset of symptoms (48-72 hrs). levels normal in 2-3days.
- Persistence of increased levels >10days denote complication like cyst,abscess.
- Sen=85%; spec=70%
- salivary gland disease and gut perforation or infarction is excluded;
- 5%cases no increase value
- Macroamylasemia
- Lipemic sample

# *Marker of pancreatitis injury*

- **Serum lipase**
  - 2 times the normal ( 2.3-20.0 IU/L)
  - n=3-5days
  - **Increased sensitivity in alcohol-induced pancreatitis;**
  - **more specific and sensitive than amylase for detecting acute pancreatitis.**
  - High levels in renal failure
- 
- **CRP**
  - **LDH**
  - **Serum Neutrophil –elastase,IL-6, and alpha macroglobulin**
  - **Serum Immunoreactive Trypsin**
  - **Triglyceride >150mg/dl**

## *Peritoneal /Plural Fluid:*

**Amylase > 5000 IU/l**

**Elevated Albumin > 30 g/L**

*Marker of biliary tract involvement*

### ➤ **Alanine aminotransferase (ALT )**

**Elevate in gallstone pancreatitis**

## *Markers of Necrosis*

**- C-reactive protein >120 mg/L**

**( Predictive of severity- Late marker-prognostic )**

**High levels associated with pancreatic necrosis**

- ❑ PMN-Elastase>120mg/L
- ❑ PLA>15U/L
- ❑ PLA<sub>2</sub>>3.5U/L

# How to Evaluate Severity?

■ RANSON CRITERIA

■ IMRIES CRITERIA

■ APACHE scoring

■ GLASGOW Criteria

■ ATLANTA score

Prediction of complication	Apache	Ranson	Glasgow
Few hours	More accurate	Less	Less
48hrs	88%	69%	84%
72 hrs	+++	++	++
Dying pt	Rising	Falling	Falling

# GLASGOW CRITERIA

- Any time during First 48hrs after admission
  - ❖ Age >55 yrs
  - ❖ 1.WBC >15000 Cu/mm
  - ❖ 2.Blood glucose>10mmol/l
  - ❖ 3.BUN >16mmol/L
  - ❖ 4.Art po<sub>2</sub>, < 60mmHg
  - ❖ 5.Ser ca. <8.0 mg/dl
  - ❖ 6.Ser Albumin<32gm/l
  - ❖ 7.Ser LDH >600u/L(n=250)
  - ❖ 8.AST Or ALT >200u/l

# RANSON CRITERIA

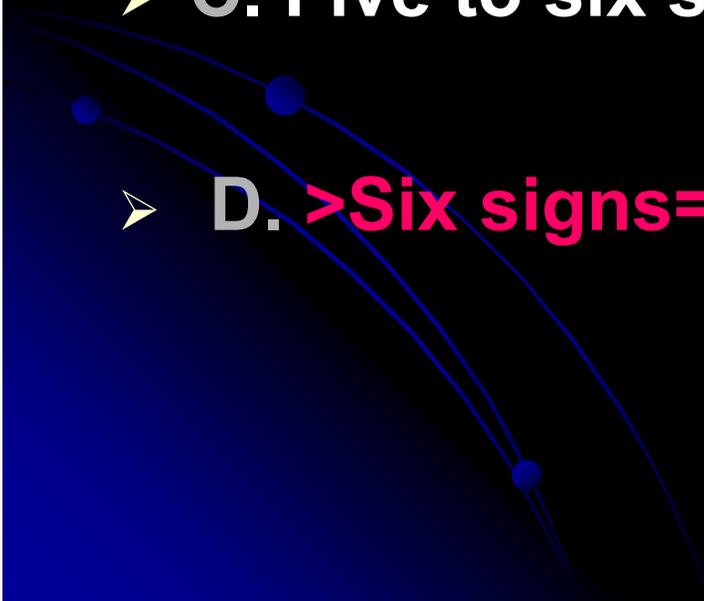
- Initial 24 hrs

- 1. Age >55 years
- 2. Glucose > 200 mg/dl
- 3. WBC > 16,000 cu/ml
- 4. LDH >350 IU/liter
- 5. AST >250 IU/liter

- Subsequent 48 hrs

- 1. Art  $O_2$  tension <60mmHg
- 2. BUN Increase >8mg/dl
- 3. Ca < 8mg/dl
- 4. Base deficit >4meq/liter
- 5. Estimated fluid sequestration >6liters
- 6. Fall in Hct >10%

# Mortality prediction (as per Ranson criteria)

- A. < 3 signs = 1%
  - B. Three to Four signs=11%
  - C. Five to six signs=33%
  - D. >Six signs= 100%
- 

# Treatment.

Mild Acute Pancreatitis

Supportive Care

- I.V. fluid resuscitation
- Nutritional support
- Analgesia

**Anti-inflammatory & Antisecretory agents drug**

Severe Pancreatitis  
(pancreatic necrosis or infected)

Antibiotic iv

➤ If not improve

Fine needle aspiration  
(FNA )

➤ If infected pancreatic

necrosis  
Percutaneous Drainage

**Thank You**

