

UNIVERSITY OF THE EAST  
RAMON MAGSAYSAY MEMORIAL  
MEDICAL CENTER, INC.  
College of Medicine

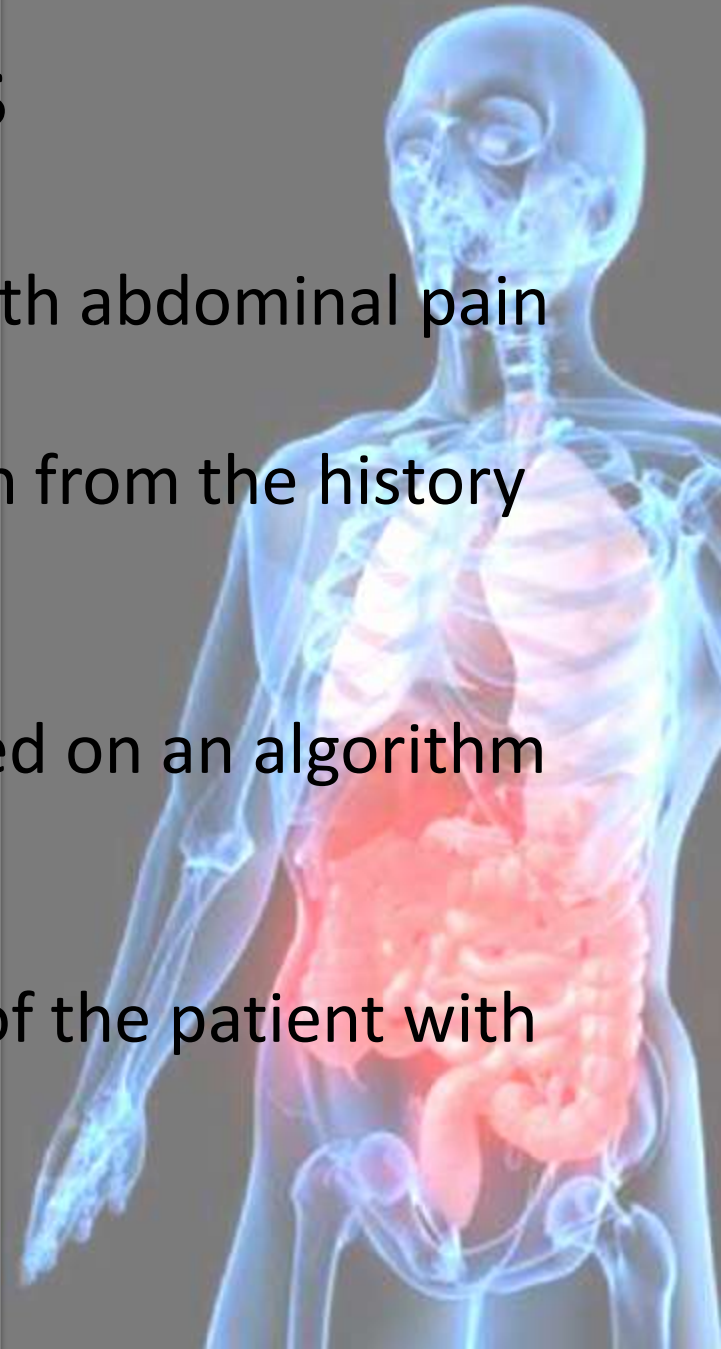
*“What’s Up, Panc?”*  
JI GRAND ROUNDS

LAGADE-ONG



# Objectives

1. To present a case of a patient with abdominal pain
2. To identify pertinent information from the history and physical examination
3. To formulate an impression based on an algorithm on abdominal pain
4. To correlate the clinical picture of the patient with supportive diagnostic exams



# Identifying Data

The patient is J.G.

- 41 y/o, female
- Filipino, Roman Catholic
- Sta. Ana, Manila
- 2<sup>nd</sup> admission in UERM
- Admitted last: November 4, 2011



# Patient Profile

- Housewife
- Preference for fatty foods
- Non-smoker
- No history of alcohol intake
- No history of illicit drug use
- Household chores as a form of exercise



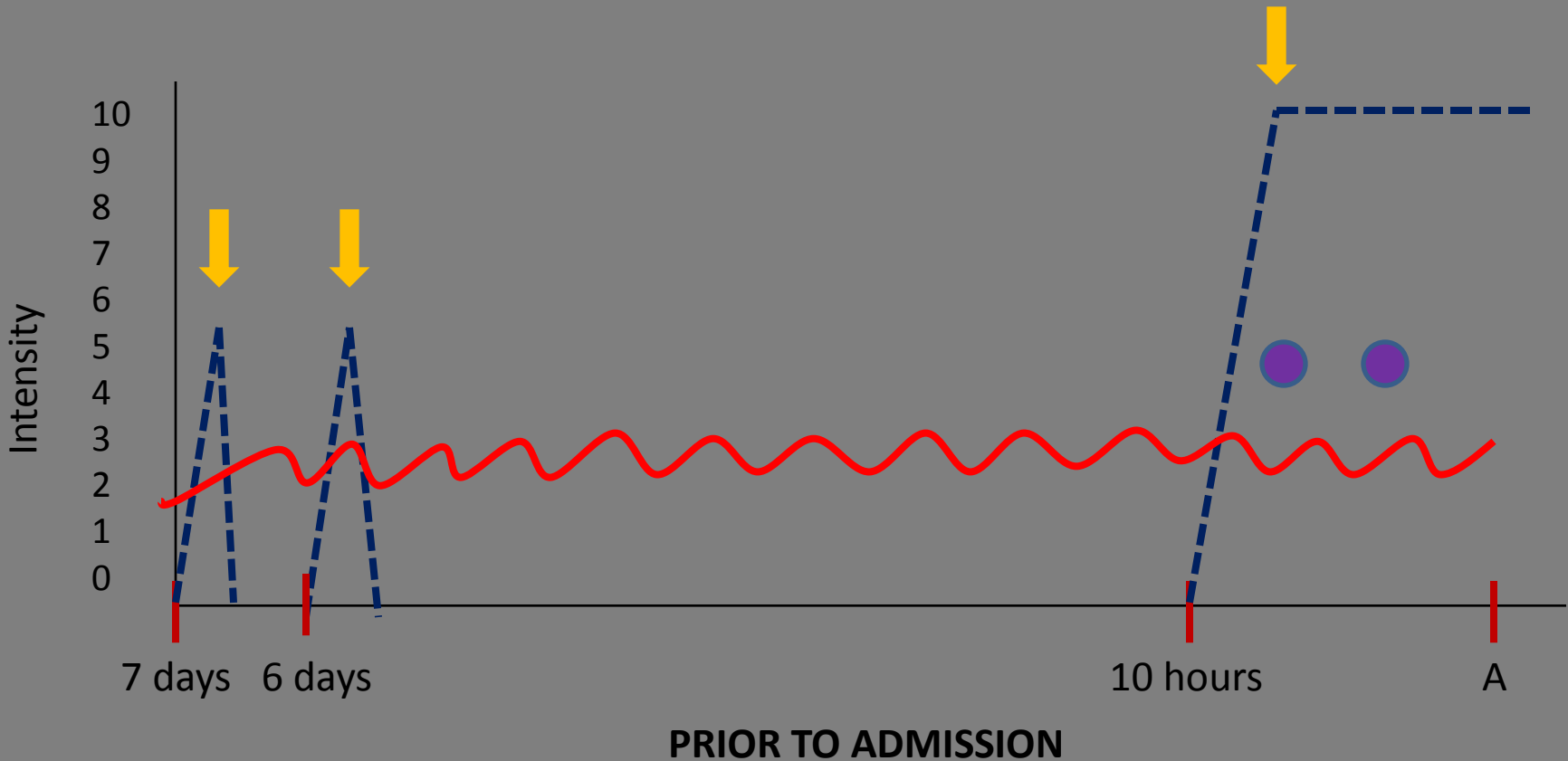
# Chief Complaint

Epigastric pain of 10  
hours duration

*Source & reliability:* Patient with good reliability



# Temporal Profile



--- Epigastric pain with radiation to the back

— Bloating & frequent flatulence



Intake of HNBB & Ranitidine

● Vomiting

# Pertinent Symptoms



POSITIVE	NEGATIVE
Epigastric pain piercing to the back	Fever
Nausea	Jaundice/icteric sclerae
Vomiting – non-bloody, non-bilious	Weight loss/malnutrition
Relieved by fetal position / aggravated by lying flat on bed	Acholic stools
Bloatedness & frequent flatulence	Change in caliber of stools
History of acute pancreatitis (2003)	Steatorrhea
Initially relieved by anti-spasmodic & H2- receptor blocker	Pruritus
	Tea-colored urine
	Melena/hematochezia/hematemesis

# Review of Systems

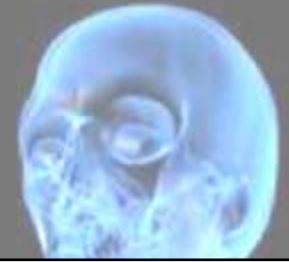


<b>GENERAL</b>	No fatigue, no sweating, <b>no weight loss</b> , no weakness
<b>SKIN</b>	No rashes
<b>EYES</b>	No changes in vision
<b>EENT</b>	No changes in hearing, no nasal discharges, no history of sore throat, no frequent colds/cough, no neck mass, no voice hoarseness, no gum bleeding
<b>RESPIRATORY</b>	No history of difficulty of breathing, <b>no hemoptysis</b>
<b>CARDIOVASCULAR</b>	No history of palpitation, no syncope, <b>no chest pain</b> , no edema, no orthopnea
<b>GASTROINTESTINAL</b>	<b>No dysphagia</b> , no changes in appetite, no indigestion, <b>no heartburn, no hematemesis, no melena</b>





# Review of Systems



## GENITOREPRODUCTIVE

LMP: 10/31/11

M: Menarche at 13 y/o, regular monthly interval, 2-3 days in duration, 2-3ppd, no dysmenorrhea

O : G2P2 (2002)

G1 – 1993, via NSD at Fabella Hospital, no fetomaternal complications

G2 – 2000, via NSD at Fabella Hospital , no fetomaternal complications

G : No STI, **No PID**, No AUB, With history of UTI – resolved

S : Coitarche at 28 y/o, 1 sexual partner, No PCB, No dyspareunia

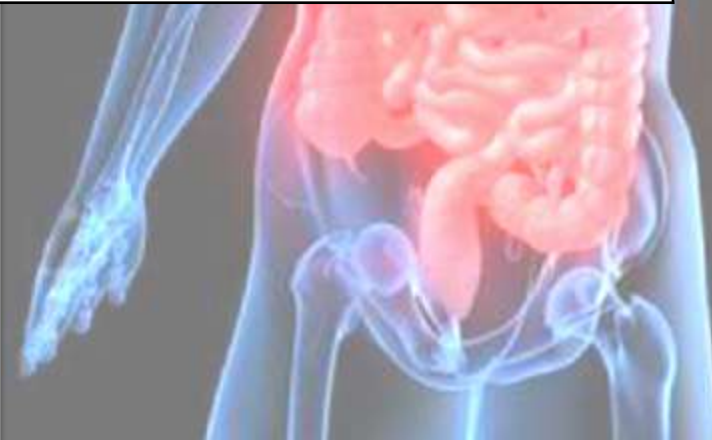
C : **No contraceptive use**



# Review of Systems

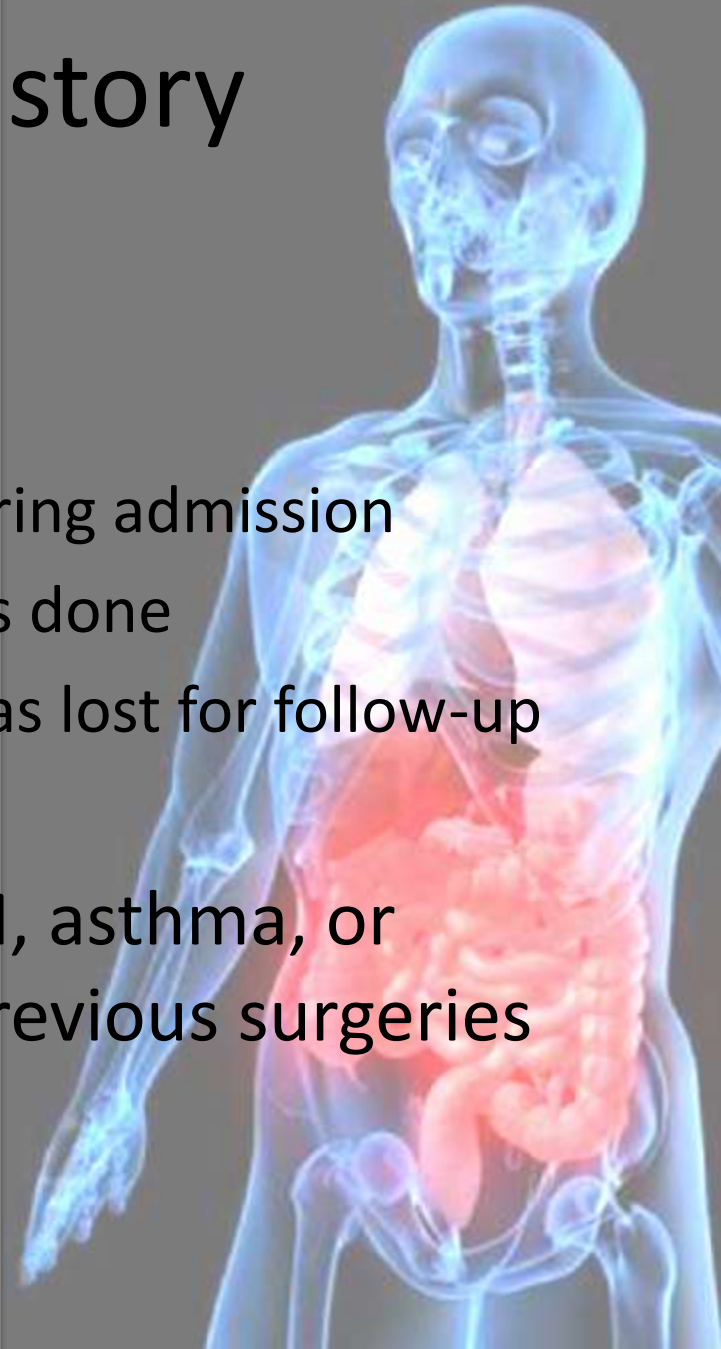


<b>BREAST</b>	No lump, no pain, no discharges
<b>EXTREMITIES</b>	No cyanosis, no clubbing, no edema
<b>HEMATOPOIETIC SYSTEM</b>	No excessive bleeding or easy bruisability
<b>NERVOUS SYSTEM</b>	No headaches, no tremors, no head trauma
<b>MUSCULOSKELETAL</b>	No joint stiffness, no swelling, no muscle weakness
<b>ENDOCRINE SYSTEM</b>	No heat/cold intolerance, no polyuria, no polydypsia, no polyphagia
<b>PSYCHIATRIC</b>	No behavioral changes



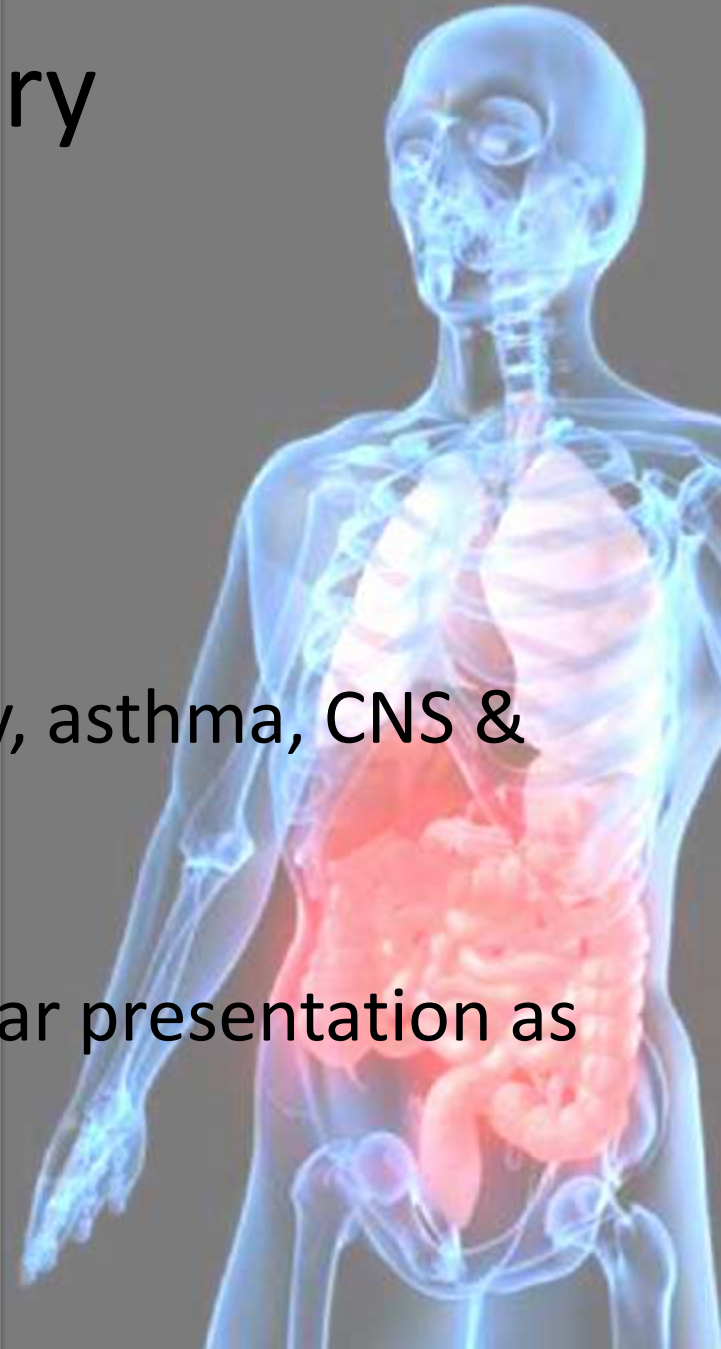
# Past Medical History

- **2003: Acute Pancreatitis**
  - Admitted in UERM for 4 days
  - Unrecalled medications given during admission
  - Unrecalled laboratory diagnostics done
  - No home medications given & was lost for follow-up
- No history of hypertension, DM, asthma, or allergy, accidents, trauma, or previous surgeries



# Family History


- Hypertension (father)
- Prostate CA (brother)
- No family history of DM, allergy, asthma, CNS & renal diseases
- No known GIT diseases or similar presentation as the patient



# Admitting Physical Examination



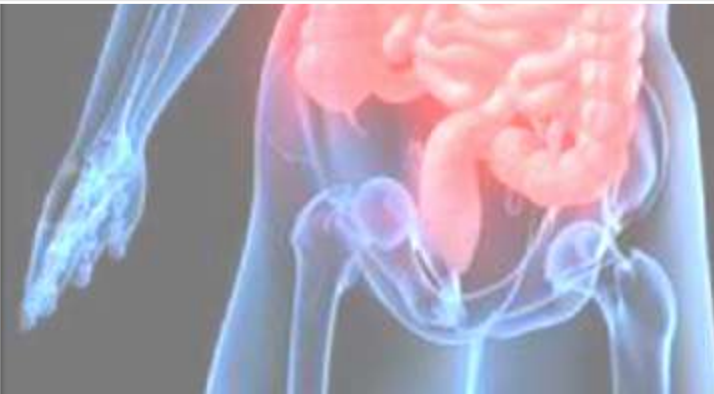
General Survey	Awake, alert, <b>in pain</b> , well-nourished
Vitals	BP: <b>120/80 mmHg</b> HR: <b>72 bpm</b> RR: <b>24 cpm</b> Temp: <b>35.0°C</b> Weight: <b>59kg</b> / Height: <b>157.5cm</b> BMI = <b>24</b>
HEENT	<b>Anicteric sclerae</b> , pink palpebral conjunctivae, 2-3mm EBRTL, full extra ocular movement, no neck vein distention, no tonsillopharyngeal congestion, <b>no cervical lymphadenopathies</b>



# Admitting Physical Examination



Chest and Lungs	No retractions, no chest lag, equal chest expansion, resonant on all lung fields, clear breath sounds on all lung fields
Heart	Adynamic precordium, normal rate and regular rhythm, distinct S1 and S2, no murmurs



# Admitting Physical Examination



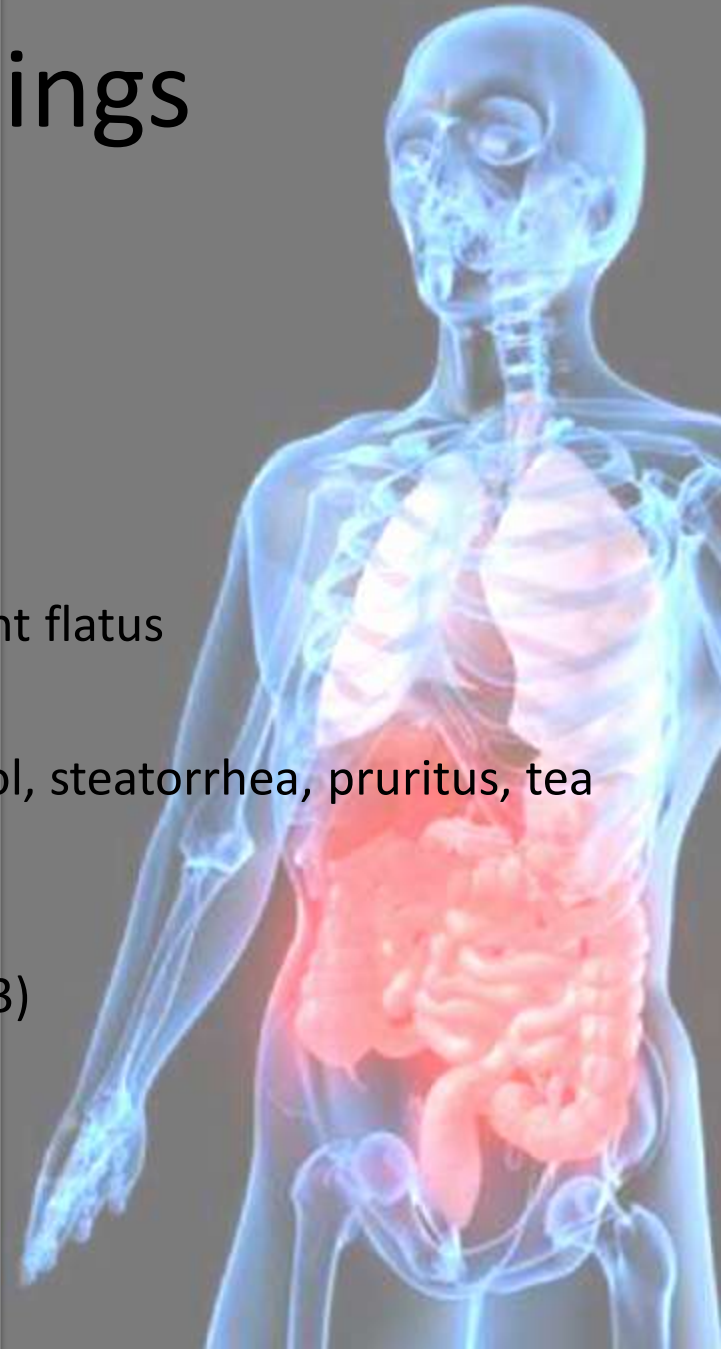
Abdomen	Flabby, <b>hypoactive bowel sounds</b> , no abdominal bruits, <b>soft</b> , tympanitic on all quadrants, liver span = 9cm, no splenomegaly, <b>with direct tenderness</b> on epigastric area on deep palpation, <b>no Murphy's sign, no Cullen's sign, no Grey Turner's sign</b> , no fluid wave
Extremities	Full range of motion, full and equal pulses, no cyanosis, no edema



# Pertinent Findings

- **Subjective Data:**

- 41 year old / female
- 1 week history of epigastric pain
- Nausea, vomiting, bloatedness and frequent flatus
- No fever, jaundice, weight loss, acholic stool, steatorrhea, pruritus, tea colored urine, and bleeding
- Previous history of Acute Pancreatitis (2003)
- No history of alcohol intake





# Pertinent Findings

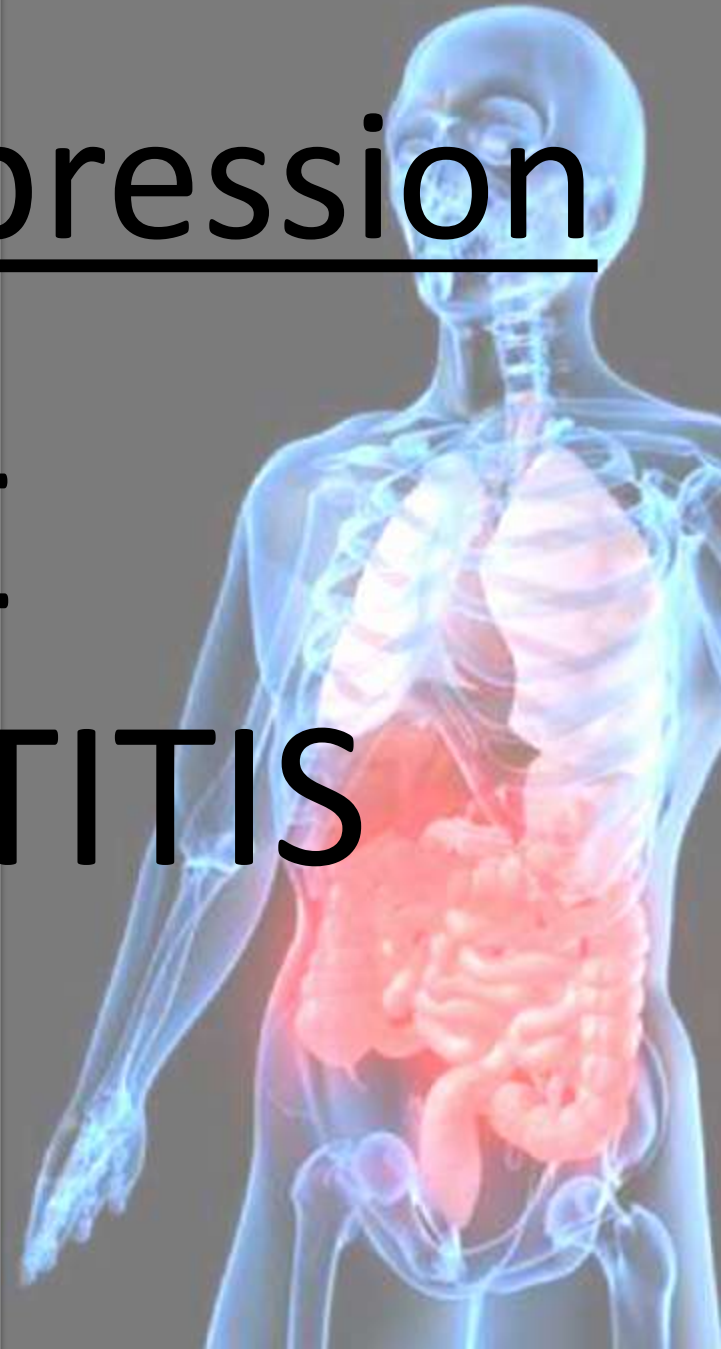
- **Objective Data:**

- Vitals: 120/80 mmHg > 72bpm > 24cpm > 35.0°C
- Anicteric sclerae
- Hypoactive bowel sounds
- Soft abdomen with direct tenderness on epigastric area on deep palpation
- No Murphy's sign, no Cullen's sign, no Grey Turner's sign, no ascitis



# Admitting Impression

## ACUTE PANCREATITIS



# Differential Diagnosis

1. Perforated Gastric Ulcer

2. Acute Cholecystitis



# APPROACH TO ABDOMINAL PAIN



Right Upper Quadrant	Epigastric	Left Upper Quadrant
Cholecystitis	Peptic ulcer disease	Splenic infarct
Cholangitis	Gastritis	Splenic rupture
Pancreatitis	GERD	Splenic abscess

Right Upper Quadrant	Epigastric
Cholecystitis	Peptic ulcer disease
Cholangitis	Gastritis
Pancreatitis	GERD
Pneumonia/empyema	Pancreatitis
Pleurisy/pleurodynia	Myocardial infarction
Subdiaphragmatic abscess	Pericarditis
Hepatitis	Ruptured aortic aneurysm
Budd-Chiari syndrome	Esophagitis

Mesenteric ischemia	Malaria	
Bowel obstruction	Familial Mediterranean fever	
Irritable bowel syndrome	Metabolic diseases	
Peritonitis	Psychiatric disease	



# Epigastric Pain

**Steady/Boring**

Acute pancreatitis, Chronic Pancreatitis, CA of Pancreas, Angina Pectoris, Dissecting Aortic Aneurysm

**Burning**

Perforated Peptic Ulcer, Angina Pectoris

**Ripping/Tearing**

Dissecting Aortic Aneurysm

**Aching**

Biliary Colic, Acute Cholecystitis

**Radiating to Back**

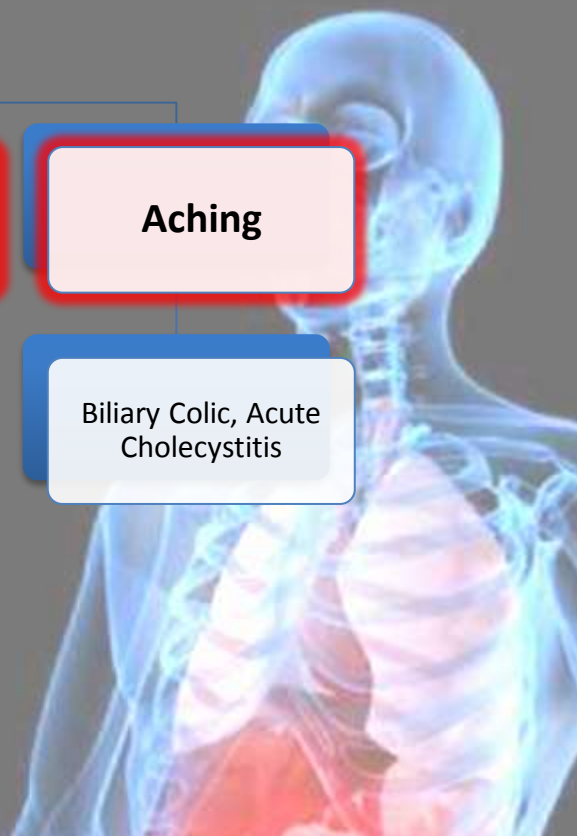
Dissecting Aortic Aneurysm, Angina Pectoris, Acute Pancreatitis

**Chronic, recurrent**

Chronic pancreatitis

**Acute, persistent**

**ACUTE PANCREATITIS**

- 
- Fauci, et.al. Harrison's Principles of Internal Medicine. (2008). USA: Mc-Graw Hill Companies Inc. 17<sup>th</sup> edition.
  - Sleisenger & Fordtrans's GI and Liver Disease (2003). Missouri: W.B. Saunders Company.
  - Bickley, L. Bates' Guide to Physical Examination and History Taking. (2007). Lippincott Williams & Wilkins. 9<sup>th</sup> edition.

Timing	Factors That May Aggravate	Factors That May Relieve	Associated Symptoms and Setting
Intermittent. Duodenal ulcer is more likely than gastric ulcer or dyspepsia to cause pain that (1) wakes the patient at night, and (2) occurs intermittently over a few weeks, then disappears for months, and then recurs.	Variable	Food and antacids may bring relief, but not necessarily in any of these disorders and least commonly in gastric ulcer.	Nausea, vomiting, belching, bloating; heartburn (more common in duodenal ulcer); weight loss (more common in gastric ulcer). Dyspepsia is more common in the young (20–29 yr), gastric ulcer in those over 50 yr, and duodenal ulcer in those from 30–60 yr.
The history of pain is typically shorter than in peptic ulcer. The pain is persistent and slowly progressive.	Often food	Not relieved by food or antacids	Anorexia, nausea, early satiety, weight loss, and sometimes bleeding. Most common in ages 50–70
Acute onset, persistent pain	Lying supine	Leaning forward with trunk flexed	Nausea, vomiting, abdominal distention, fever. Often a history of previous attacks and alcohol abuse or gallstones
Chronic or recurrent course	Alcohol, heavy or fatty meals	Possibly leaning forward with trunk flexed; often intractable	Symptoms of decreased pancreatic function may appear: diarrhea with fatty stools (steatorrhea) and diabetes mellitus.
Persistent pain; relentlessly progressive illness		Possibly leaning forward with trunk flexed; often intractable	Anorexia, nausea, vomiting, weight loss, and jaundice. Emotional symptoms, including depression
Rapid onset over a few minutes, lasts one to several hours and subsides gradually. Often recurrent			Anorexia, nausea, vomiting, restlessness
Gradual onset; course longer than in biliary colic	Jarring, deep breathing		Anorexia, nausea, vomiting, and fever
Often a gradual onset			Fever, constipation. There may be initial brief diarrhea.

# Primary impression: **ACUTE PANCREATITIS**

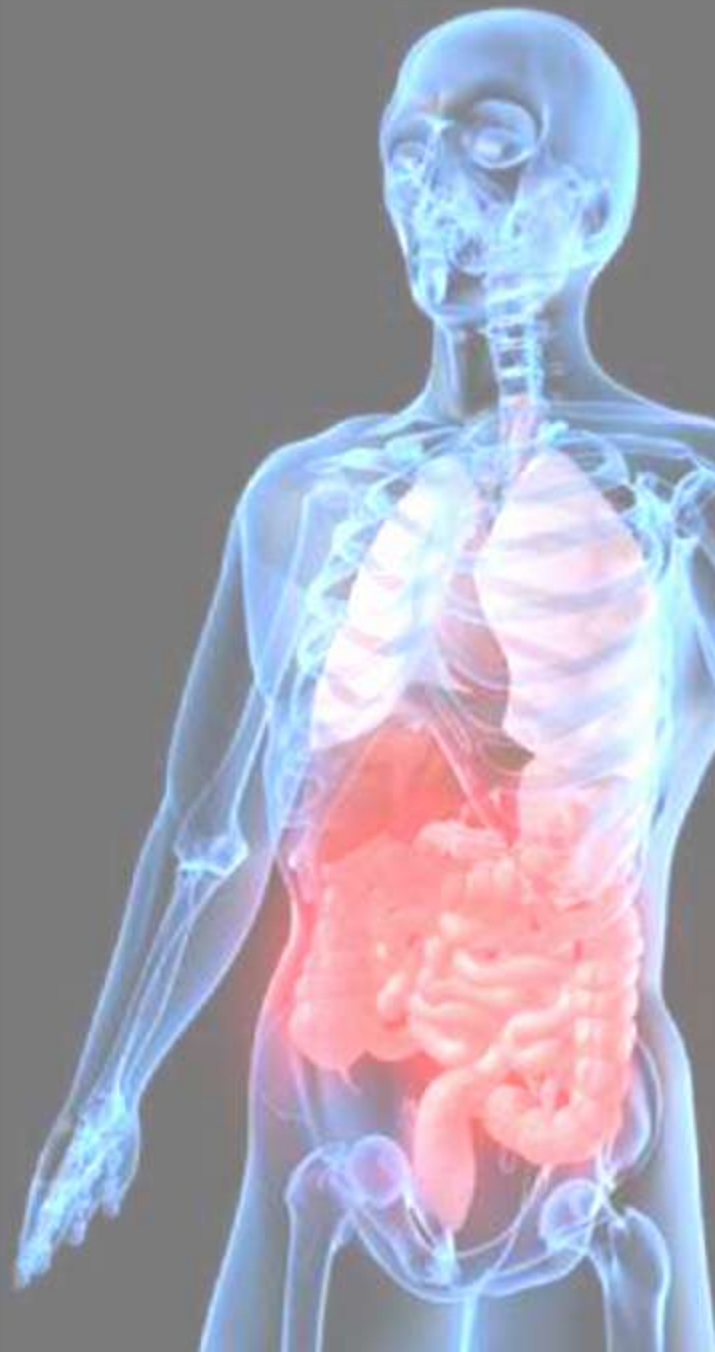


Expected Findings	Patient
Epigastric pain, often with radiation to the back.	✓
Nausea, vomiting, sweating, weakness.	✓
Abdominal tenderness	✓
Diminished or absent bowel sounds	✓
Fever	✗

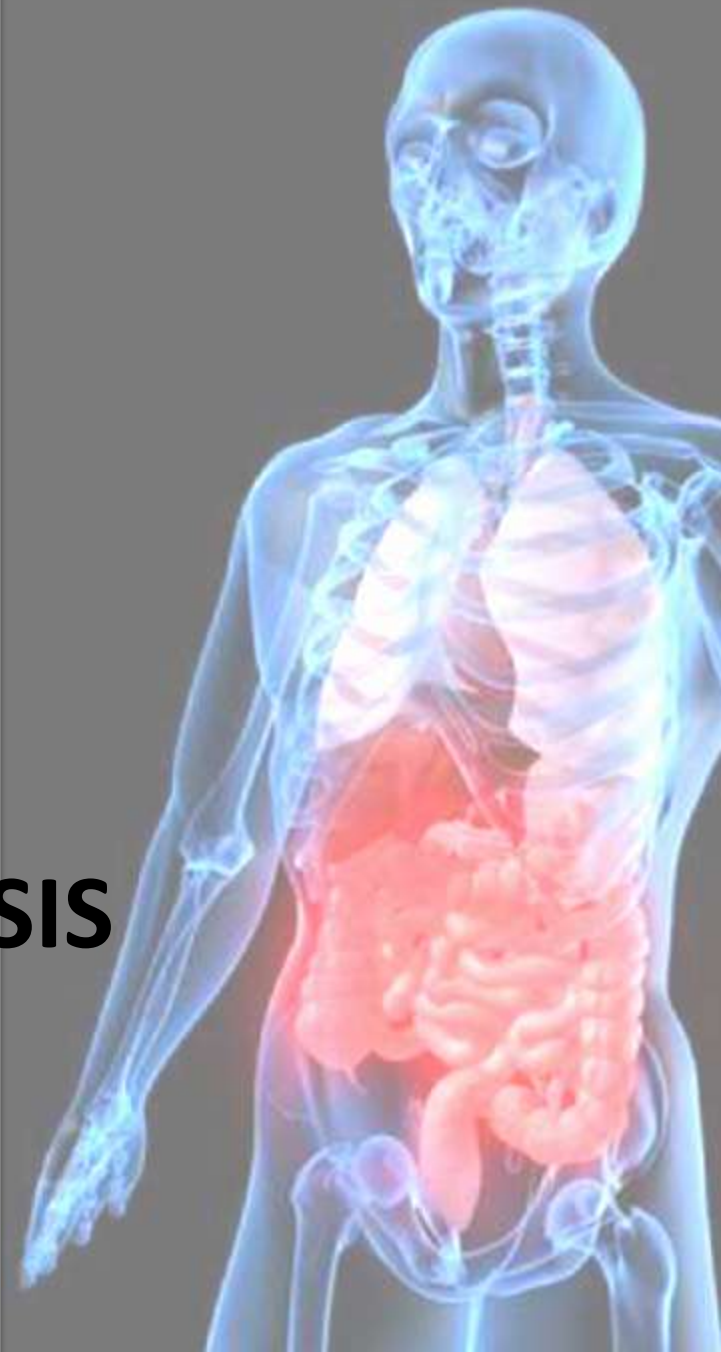




# DIAGNOSTICS

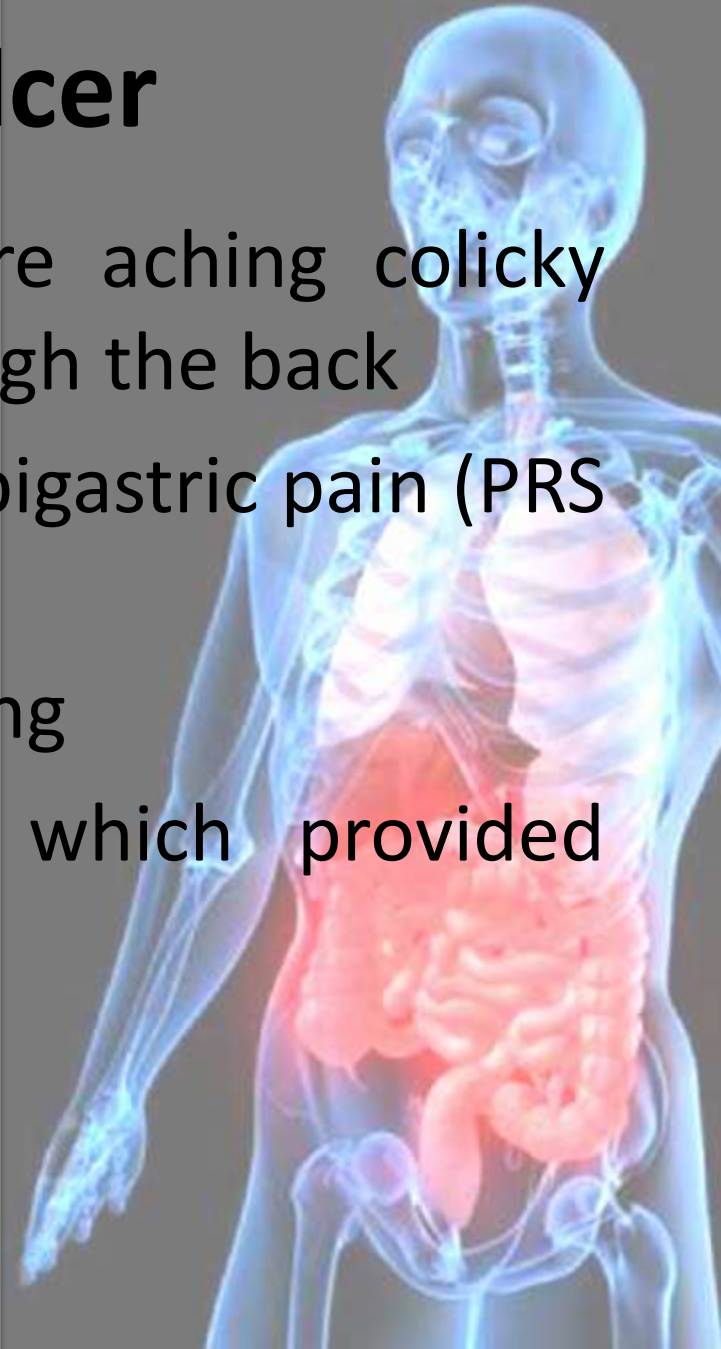


# DIFFERENTIAL DIAGNOSIS



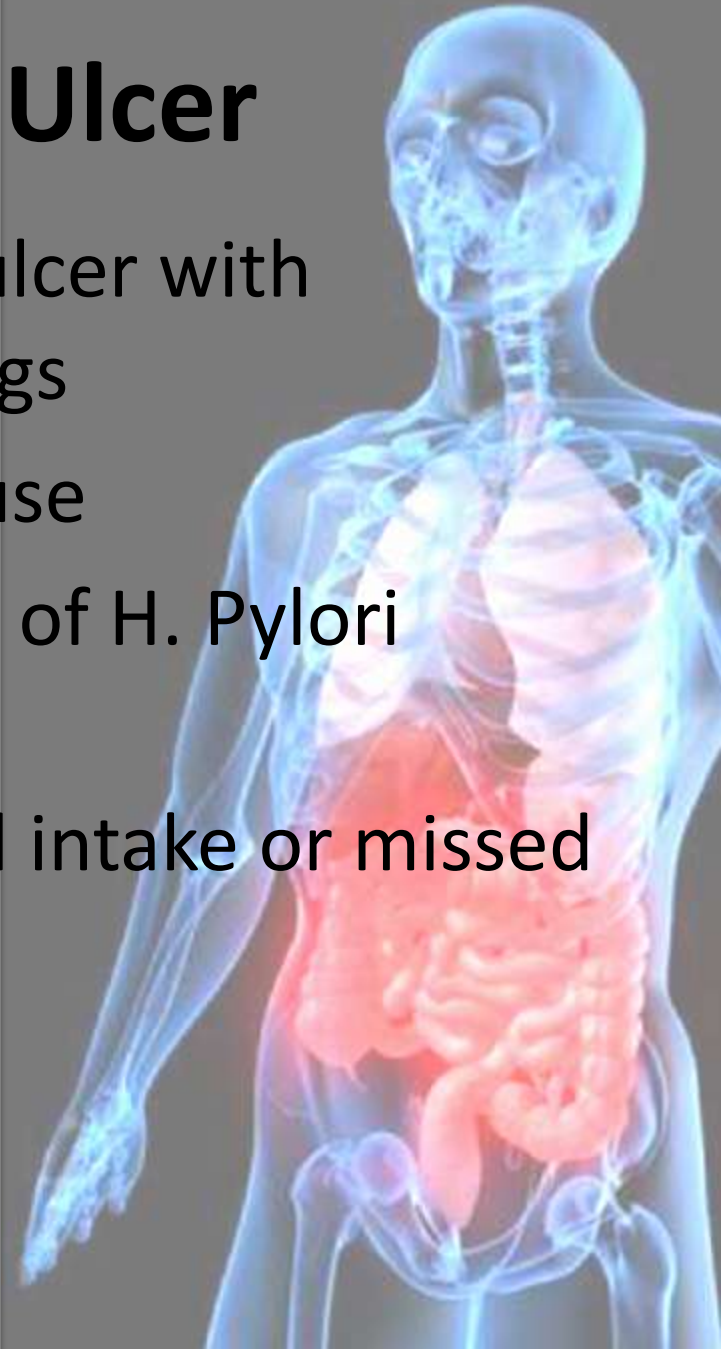
# RULE IN: Perforated Ulcer

- Sudden recurrence of severe aching colicky epigastric pain piercing through the back
- Prior episodes of tolerable epigastric pain (PRS 5-6/10) ~ 15-30 minutes
- Nausea, vomiting, and bloating
- History of Ranitidine use which provided transient relief



# RULE OUT: Perforated Ulcer

- No history of PUD or gastric ulcer with supporting endoscopic findings
- No history of chronic NSAID use
- No tests confirming presence of H. Pylori infection
- Pain not associated with food intake or missed meals



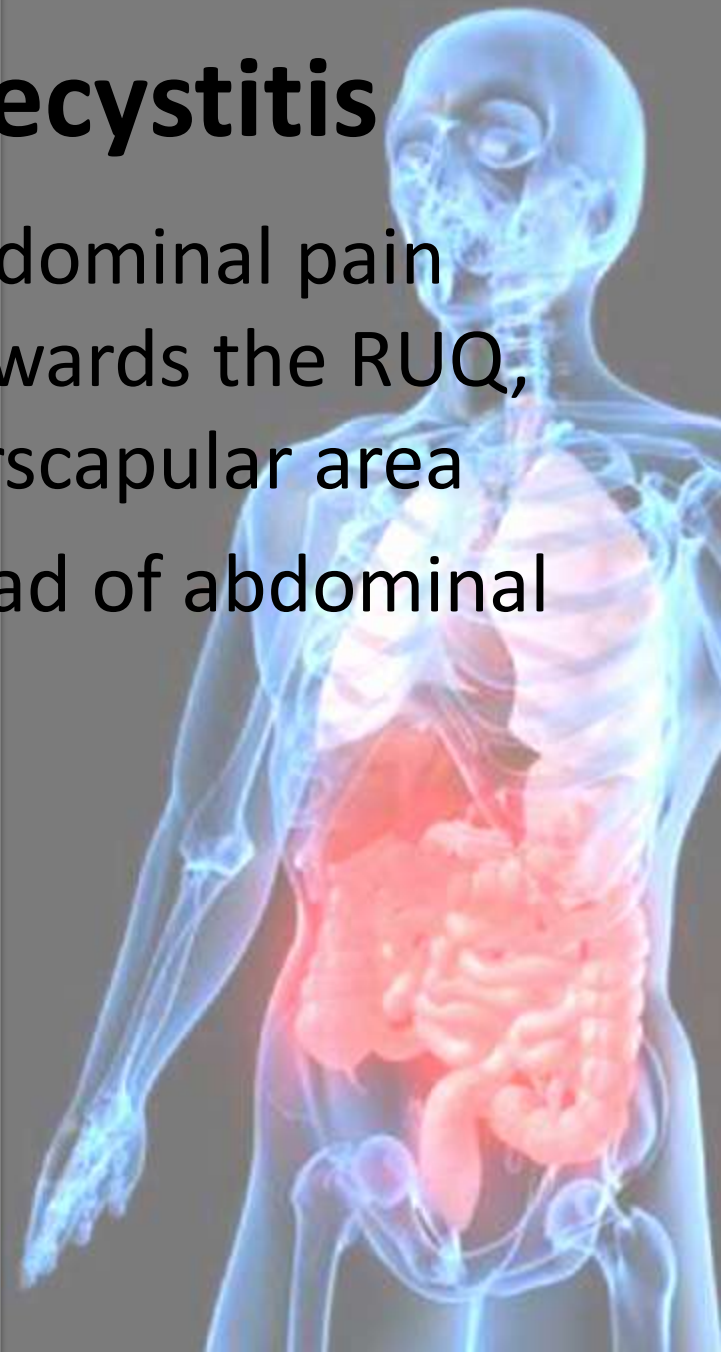
# RULE IN: Acute Cholecystitis

- Abdominal pain (epigastric)
- Leukocytosis
- Nausea, vomiting, abdominal distention, hypoactive bowel sounds
- Risk Factors: Forty, Fat, Female, Fertile/Flatulent



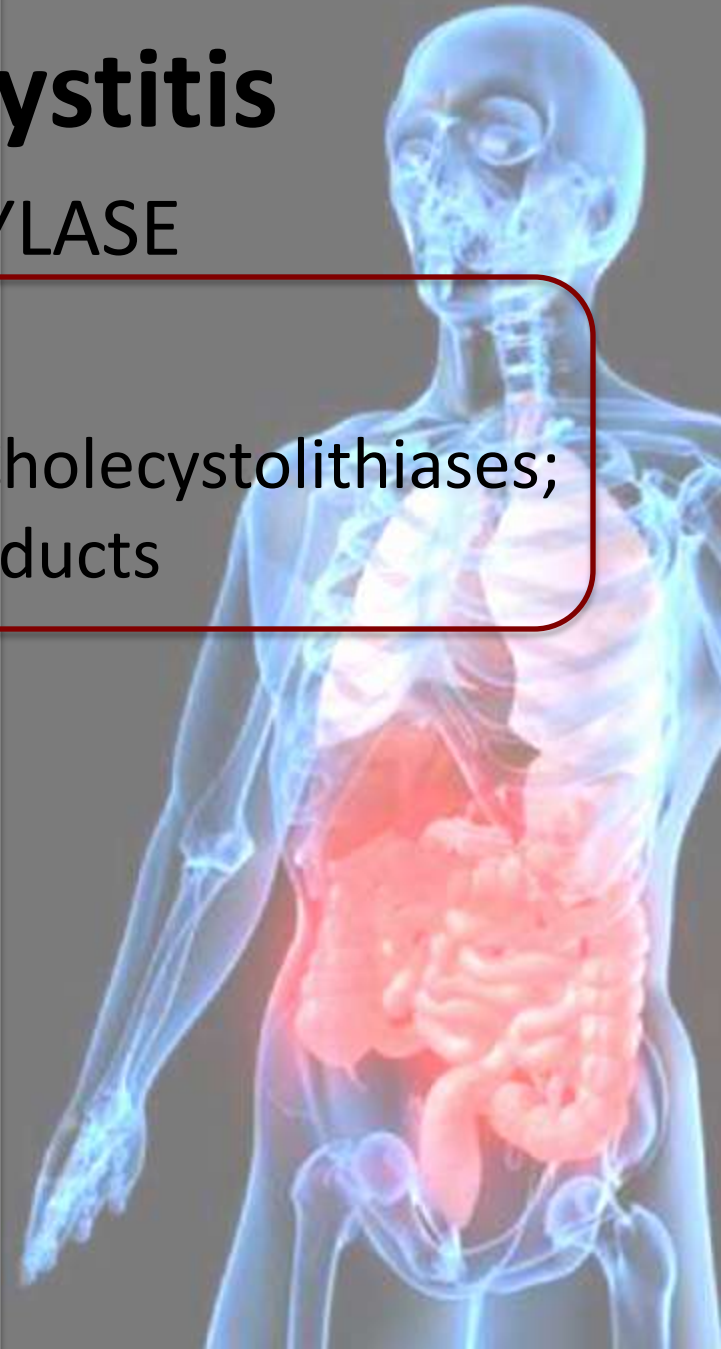
# RULE OUT: Acute Cholecystitis

- Absence of progression of abdominal pain from initial epigastric area towards the RUQ, right shoulder, and right interscapular area
- Absence of fever from the triad of abdominal pain, fever, and leukocytosis
- No Murphy's sign

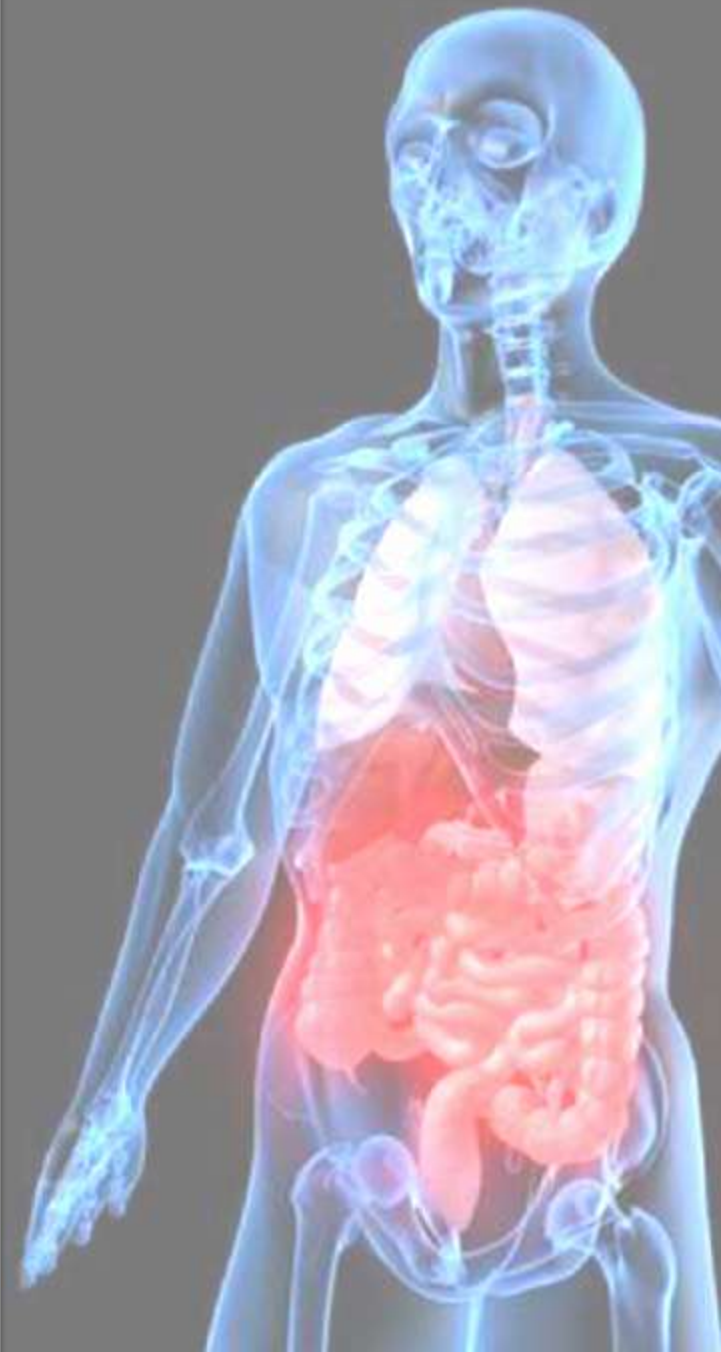


# RULE IN: Acute Cholecystitis

- Elevated levels of Serum AMYLASE
- Ultrasound findings of:
  - Fatty liver; Cholecystitis with Cholecystolithiasis;
  - Dilated CBD and intrahepatic ducts



# DISCUSSION

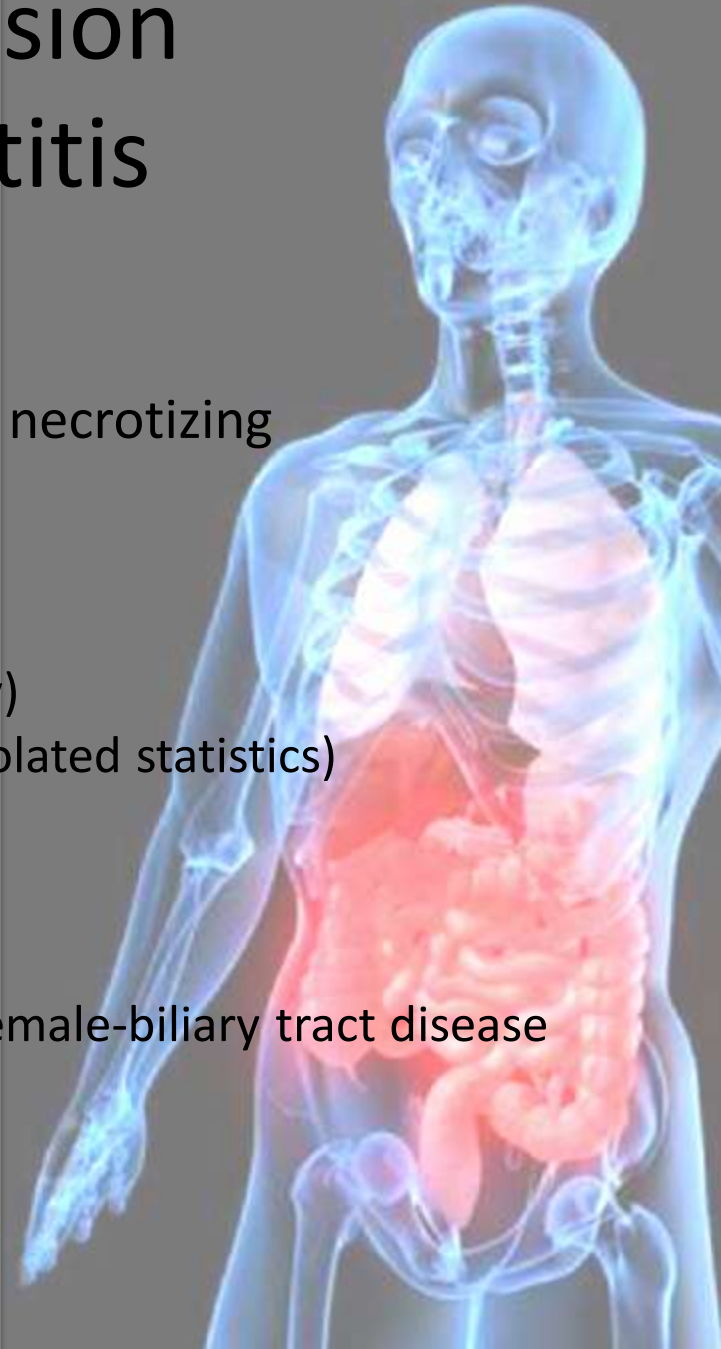




# Primary Impression

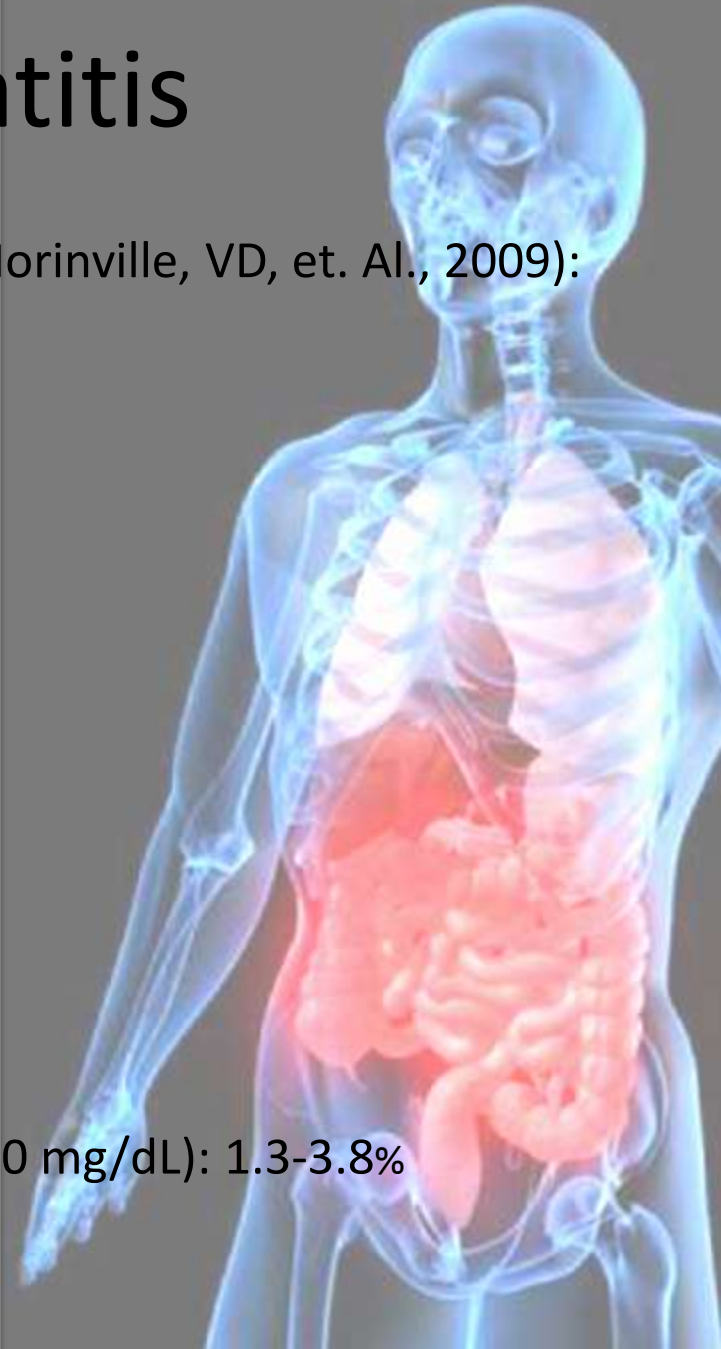
## Acute Pancreatitis

- Pancreatic inflammatory disease
- Spectrum: interstitial (mild, self-limited) → necrotizing
- Incidence varies with location & etiology
  - England: 5.4/100,000
  - US: 79.8/100,000 (>200,000 cases annually)
  - Philippines: 25, 365 cases annually (extrapolated statistics)
- Risk Factors
  - Race- 3x higher in blacks than whites
  - Sex- male predominance; Male-alcohol, Female-biliary tract disease

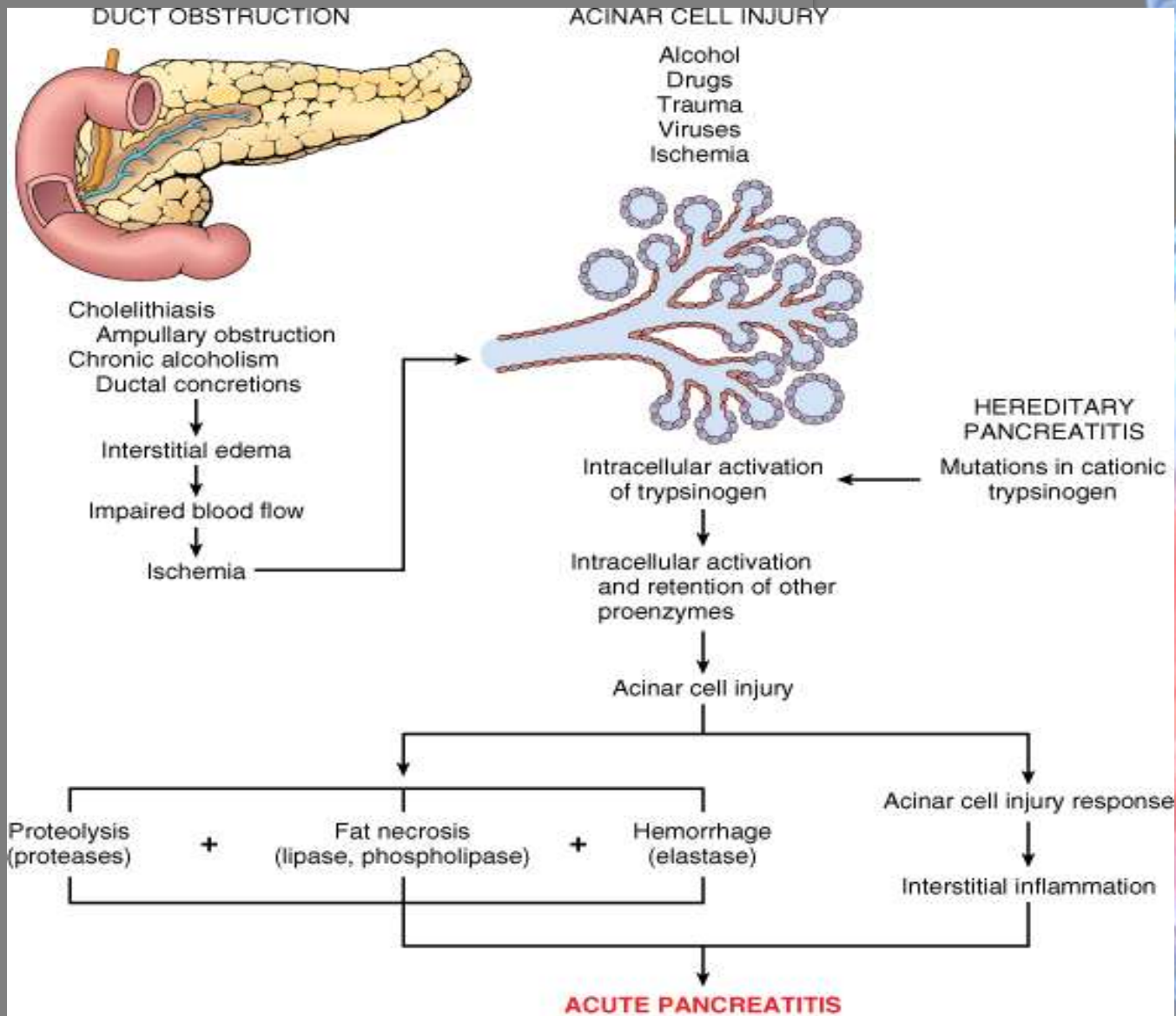


# Acute pancreatitis

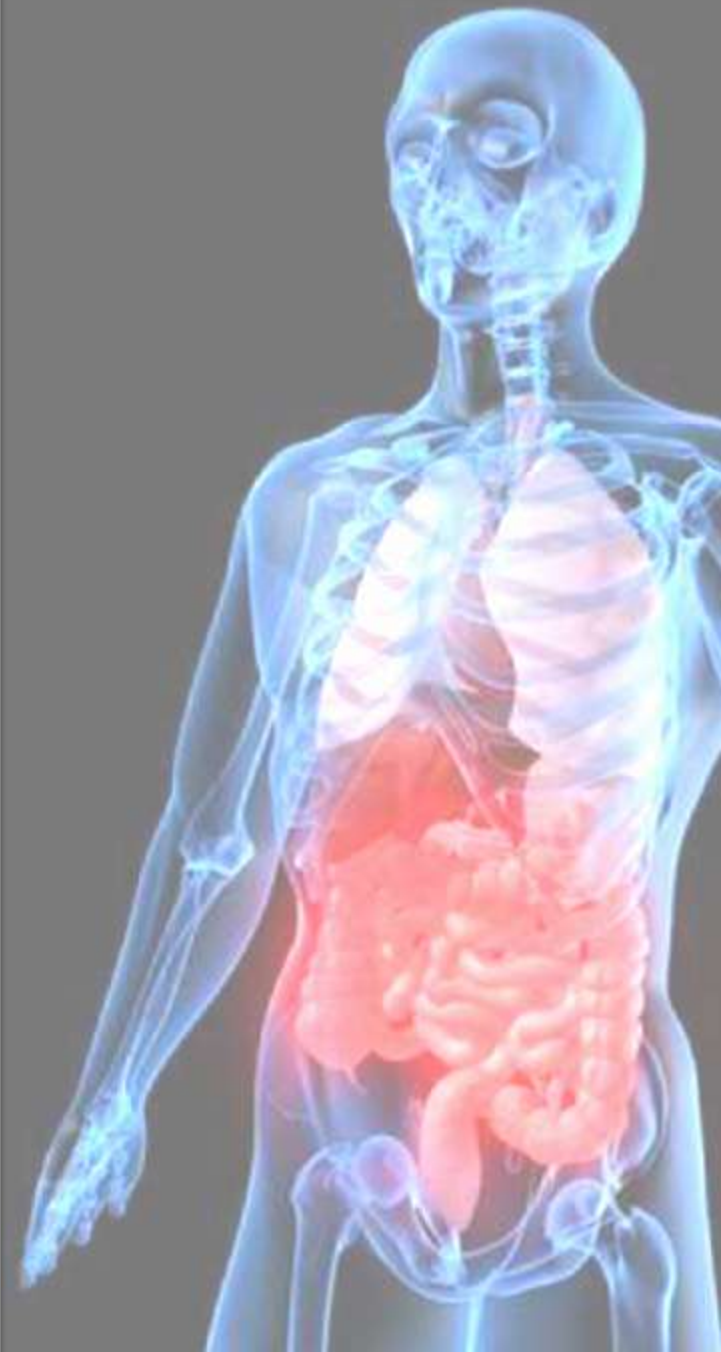
- Age
  - Median age at onset based on etiologies (Morinville, VD, et. Al., 2009):
    - **Alcohol related- 39 years**
    - **Biliary tract disease- 69 years**
    - Trauma related- 66 years
    - Drug induced- 42 years
    - ERCP related- 58 years
    - AIDS related- 31 years
    - Vasculitis related- 36 years
- Etiology
  - **Gallstones: 30-60%**
  - **Alcohol: 15-30%**
  - ERCP: 5-20%
  - Drug-related: 2-5%
  - Hypertriglyceridemia (>11/3 mmol/L or 1000 mg/dL): 1.3-3.8%



# PATHOGENESIS

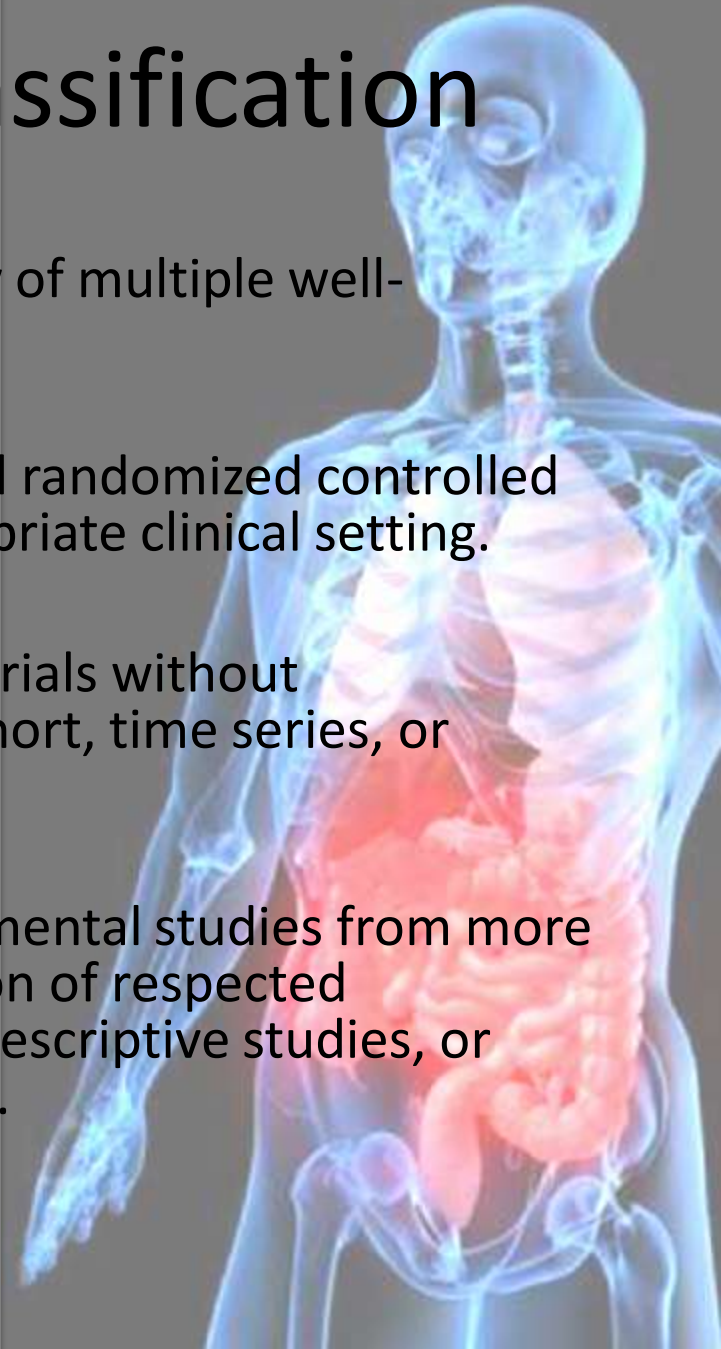


**MANAGEMENT**



# Evidence Ratings Classification

- I. At least one published systematic review of multiple well-designed randomised controlled trials.
- II. At least one published properly designed randomized controlled trial of appropriate size and in an appropriate clinical setting.
- III. Evidence from published well-designed trials without randomization, single group prepost, cohort, time series, or matched case-controlled studies.
- IV. Evidence from well-designed nonexperimental studies from more than 1 center or research group or opinion of respected authorities, based on clinical evidence, descriptive studies, or reports of expert consensus committees.

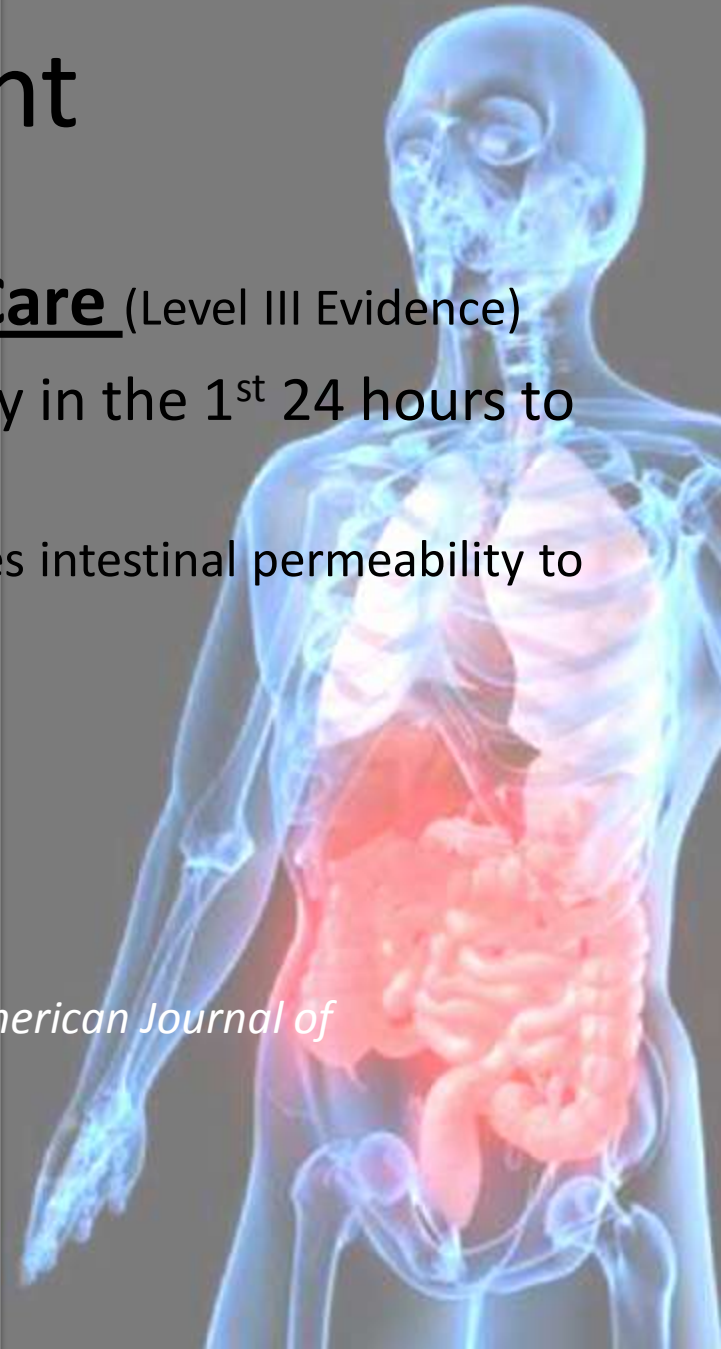


# Management

## Treatment Guideline I: **Supportive Care** (Level III Evidence)

- Fluid resuscitation- important especially in the 1<sup>st</sup> 24 hours to prevent hypovolemia.
  - Prevent intestinal ischemia (which increases intestinal permeability to bacteria).

*Practice guidelines in Acute Pancreatitis (Banks, P.A., American Journal of Gastroenterology, 2006)*



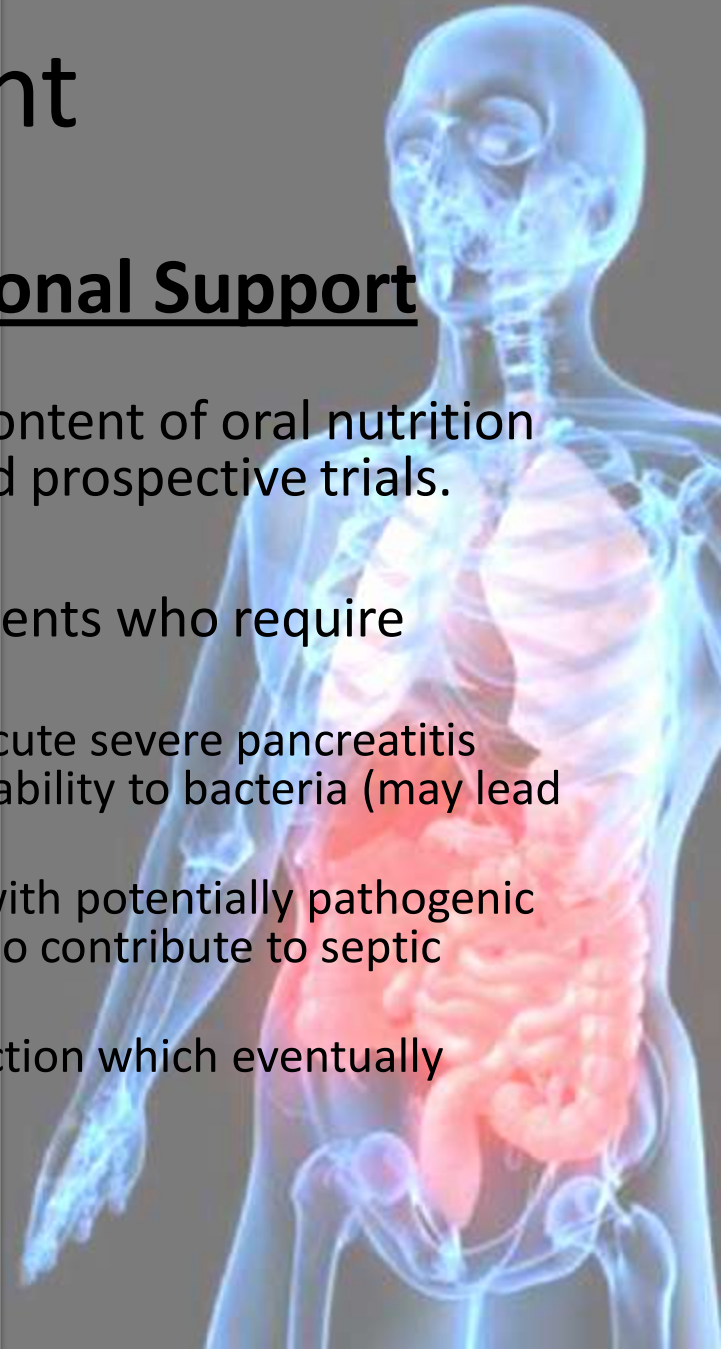
# Management

- Treatment Guideline II: **Transfer to intensive care unit**  
(level III Evidence)
  - This is imperative if there are signs that the pancreatitis is severe or going to be severe.
    - Organ dysfunction
    - Sustained hypoxemia
    - Hypotension
  - Other danger signals to be considered:
    - Obesity (BMI>30)
    - Oliguria with urine output (<50 mL)
    - Tachycardia (>120 beats/minute)
    - Evidence of encephalopathy
    - Increase need of narcotic agents to counteract pain.



# Management

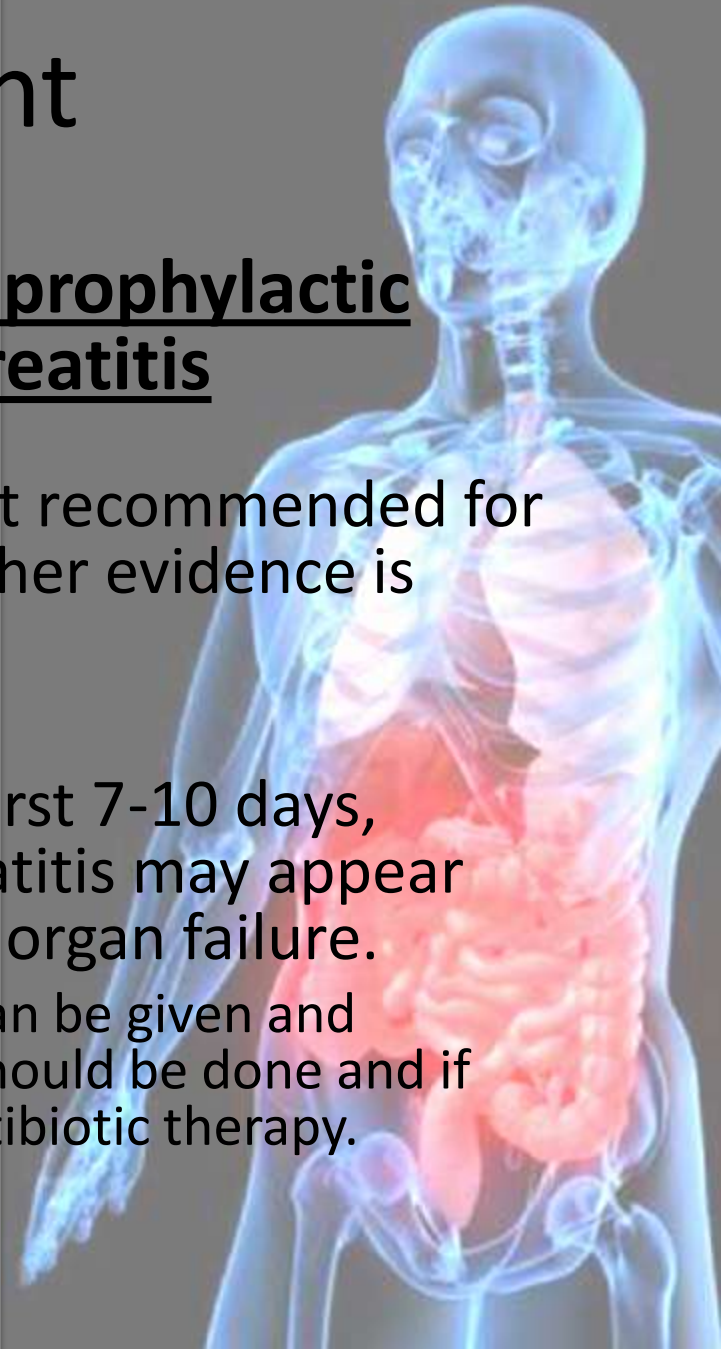
- Treatment Guideline III: **Nutritional Support**  
(Evidence Level II)
  - Exact timing of oral nutrition and the content of oral nutrition have not been subjected to randomized prospective trials.
  - Enteral feeding is still preferred for patients who require nutritional support.
    - Gut barrier function is compromised in acute severe pancreatitis which results to greater intestinal permeability to bacteria (may lead to infected necrosis)
    - Higher incidence of gastric colonization with potentially pathogenic enteric bacteria in severe disease that also contribute to septic complications.
    - Enteral Feeding stabilizes gut barrier function which eventually improves morbidity and mortality.





# Management

- Treatment Guideline IV: **Use of prophylactic antibiotics in necrotizing pancreatitis**  
(Evidence level III)
  - Prophylactic antiobiotic use is not recommended for necrotizing pancreatitis until further evidence is available.
  - It is understood that during the first 7-10 days, patients with necrotizing pancreatitis may appear septic with leukocytosis, fever or organ failure.
    - In this interval, antibiotic therapy can be given and evaluation for source of infection should be done and if found negative on all tests, stop antibiotic therapy.



# Complications

- Acute Fluid Collections
  - Pancreatic ascites
  - Pleural effusion
- Pseudocyst
- Intraabdominal infections (Pancreatic Abscess)
- Pancreatic necrosis (sterile or infected) 40-60%



# Complications: Systemic

- Shock
- GI bleeding
- CBD obstruction
- Ileus
- Splenic infarction/  
rupture
- Disseminated  
Intravascular Coagulation
- Subcutaneous fat  
necrosis
- ARDS
- Pleural effusion
- ARF
- Sudden blindness



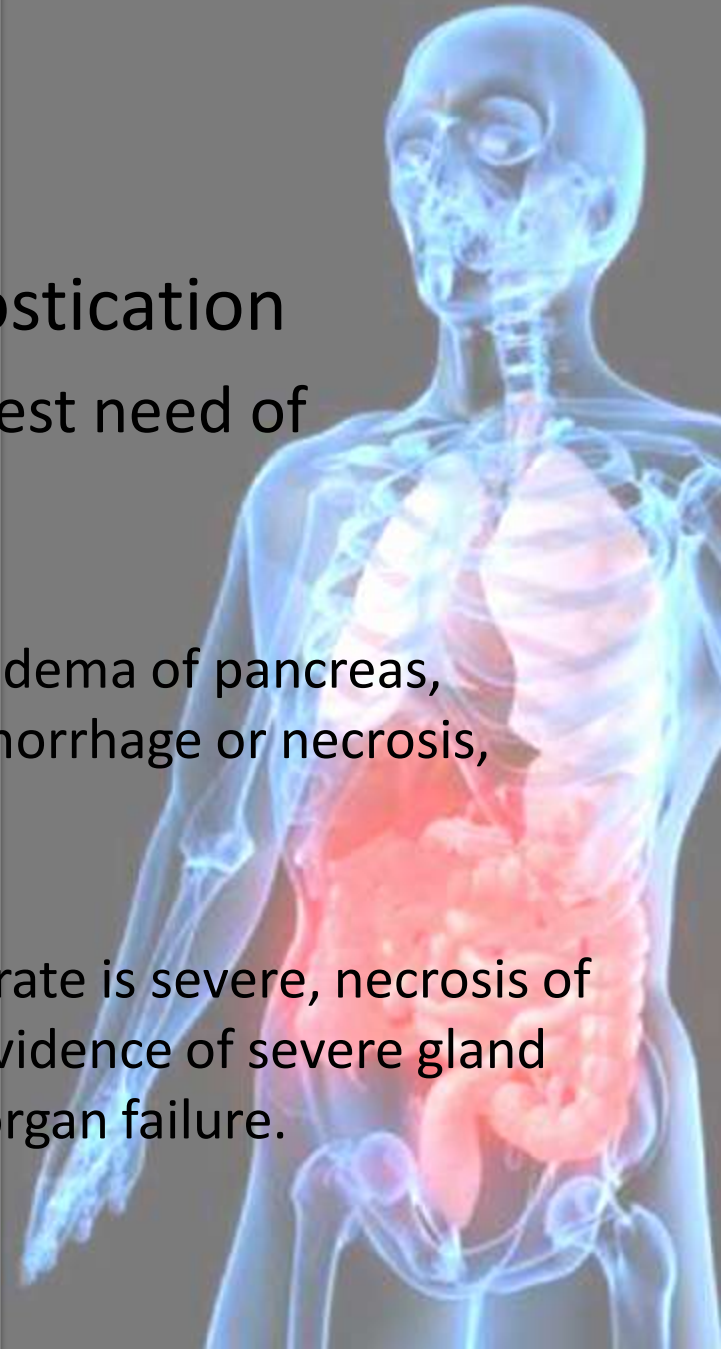
# Prognosis

- Risk factors that adversely affect survival of severe acute pancreatitis
  - Severe Acute Pancreatitis
    1. Associated with organ failure and/or local complications such as necrosis
    2. Clinical manifestations
      - a. Obesity BMI > 30
      - b. Hemoconcentration (hematocrit > 44%)
      - c. Age > 70
    3. Organ failure
      - a. Shock
      - b. Pulmonary insufficiency (PO<sub>2</sub> < 60)
      - c. Renal failure (CR > 2.0 mg%)
      - d. GI bleeding
    4. Ranson criteria (not fully utilizable until 48 h)
    5. Apache II score > 8 (cumbersome)



# Prognosis

- Clinical Assessment and prognostication
  - Used to identify patients in greatest need of aggressive medical treatment
- Mild disease= can have interstitial edema of pancreas, inflammatory infiltrate without hemorrhage or necrosis, minimal or no organ dysfunction
- Severe disease= inflammatory infiltrate is severe, necrosis of the parenchyma accompanied by evidence of severe gland dysfunction associated with multi-organ failure.



# Ranson Criteria

Criteria	On admission		Criteria	After 48 hours	
	Patient	Score		Patient	Score
Age (> 55y/o)	41	0	Hct fall (> 10%)	14%	1
WBC (> 16 g/L)	19.2	1	Urea rise ( $\geq$ 5.0 mmol/L)	x	x
Glucose (>11.1 mmol/L)	8.3	0	Serum Calcium ( $\leq$ 2.0 mmol/L)	2.3	0
LDH (> 350 UI/L)	317	0	PaO <sub>2</sub> ( $\leq$ 60 mmHg)	79	0
SGOT (> 250 UI/L)	242	0	Base deficit (BE $\geq$ 4.0 mmol/L)	0.3	0
			Fluid sequestration (> 6 L)	< 6 L (no evidence of third spacing)	0
			<b>TOTAL</b> (after 48 hours)		<b>2</b>

# APACHE II



Beginning : Date ----- Time ----- APACHE II patients study number Patients initial

## Acute Physiology and Chronic health evaluation

A: Acute physiology score (12 variables)	High abnormal range					Low abnormal range			
	+4	+3	+2	+1	0	+1	+2	+3	+4
Physiological Variables	+4	+3	+2	+1	0	+1	+2	+3	+4
Temperature – rectal (°C)	≥41	39-40.9		38.5–38.9	36–38.4	34–35.9	32–33.9	30–31.9	≤29.0
Mean arterial pressure (mm Hg)	≥160	130–159	110–129		70–109		50–69		≤49
Heart rate-ventricular response	≥180	140–179	110–139		70–109		55–69	40–54	≤39
Respiratory rate non ventilated or ventilated	≥50	35–49		25–34	12–24	10–11	6–9		≤5
Oxygen: A – a DO or PaO <sub>2</sub> (mm Hg) FiO <sub>2</sub> ≥ 0.5 record A – aDO <sub>2</sub> FiO <sub>2</sub> < 0.5 record only PaO <sub>2</sub>	≥500	350-499	200–349		<200 PO <sub>2</sub> >70	PO <sub>2</sub> 61–70		PO <sub>2</sub> 55–60	PO <sub>2</sub> <55
Arterial pH	≥7.7	7.6–7.69		7.5-7.59	7.33–7.49		7.25–7.32	7.15–7.24	<7.15
Serum HCO <sub>3</sub> – only if no ABGs	≥52	41.5–1.9		32–40.9	23–31.9		18–21.9	15–17.9	<15
Serum sodium (mmol/l)	180	160–179	155–159	50–154	130–149		120–129	111–119	≤110
Serum potassium (mmol/l)	≥7	6–6.9		5.5–5.9	3.5–5.4	3–3.4	2.5–2.9		<2.5
Serum creatinine (umol/l)	≥350	200–340	150–190		60–140		<60		
Haematocrit (%)	≥60		50–50.9	46–49.9	30–45.9		20–29.9		<20
White Blood cell court (x1000 /mm <sup>3</sup> )	≥40		20–39.9	15–19.9	30–14.9		1–2.9		<1
Glasgow Coma Score (GCS)	Score = 15 minus actual GCS								

**[Table/Fig-1]:** The APACHE II chart for scoring



### B. Age points

Age years	Points	History	Points for elective surgery	Points for emergency surgery and non-operative patients	Apache II score: sum of A + B + C
≥ 44	0	Liver: Biopsy proven cirrhosis and documented portal hypertension or prior episodes of hepatic failure	2	5	A: APS score
45-54	2	Cardiovascular NYHA class IV	2	5	B: Age Points score
55-64	3	Respiratory eg. Severe COPD, hypercapnia, home O2 pulmonary hypertension	2	5	C: Chronic health points score
65-74	5	Renal chronic dialysis	2	5	
≥ 75	6	Immunocompromised	2	5	Total apache II

[Table/Fig-2]: The APACHE II chart for scoring

**PATIENT'S SCORE: 2 Pts**



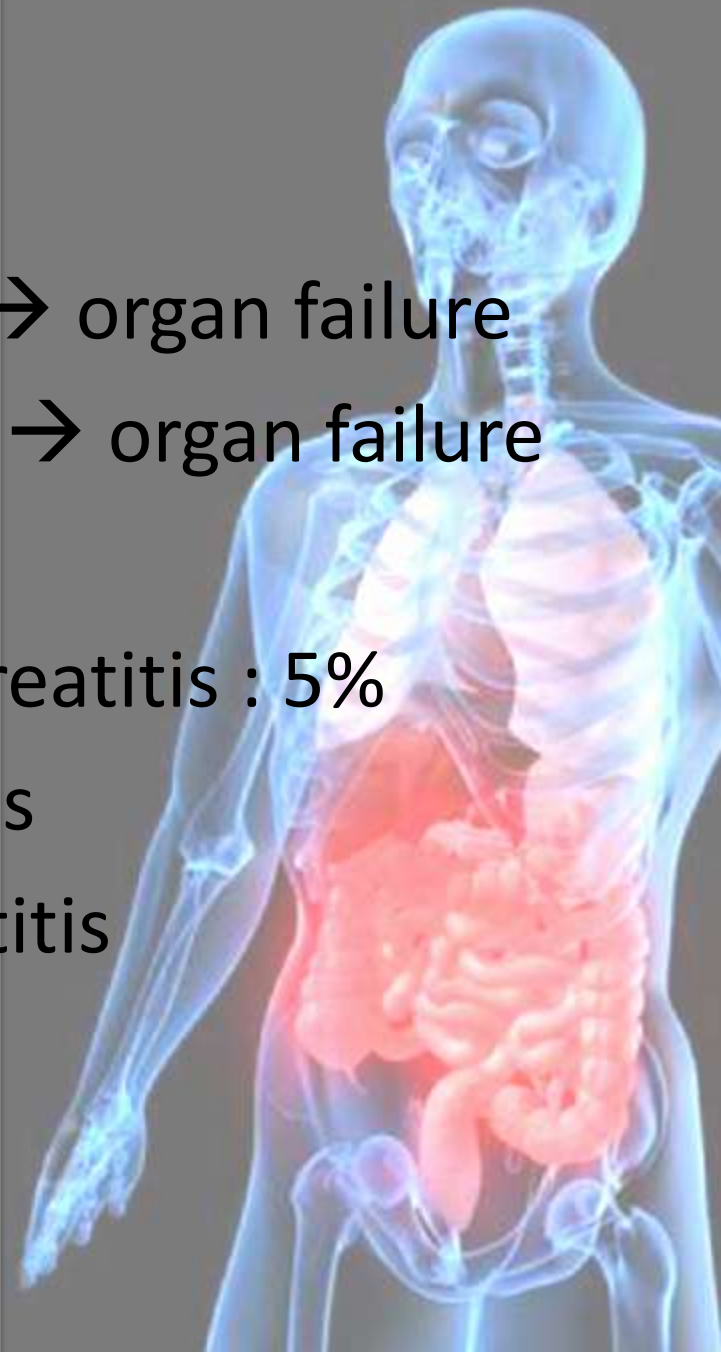


# Prognosis

10% of interstitial pancreatitis → organ failure  
54% of necrotizing pancreatitis → organ failure

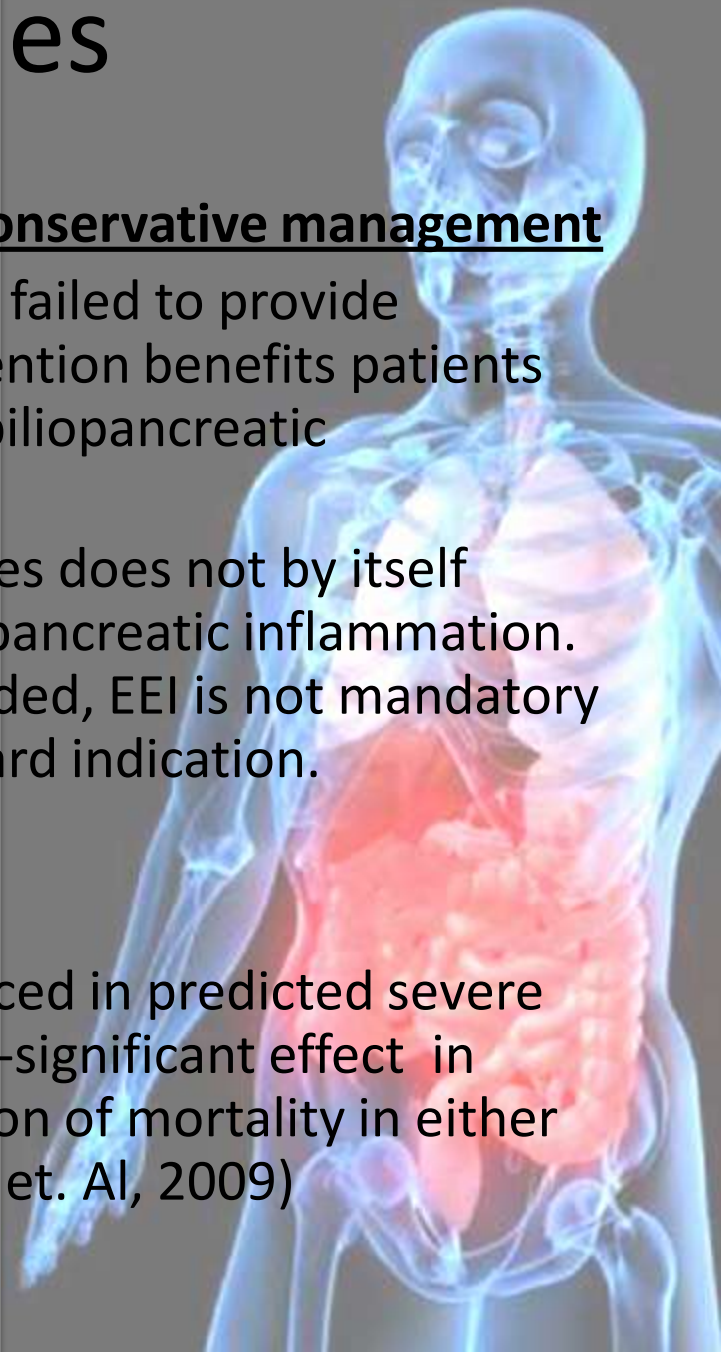
Overall mortality in acute pancreatitis : 5%

- \* 3% in interstitial pancreatitis
- \* 17% in necrotizing pancreatitis



# Recent Studies

- **Early endoscopic intervention vs. Early Conservative management**
  - In a study conducted by Oria, A. et. Al. failed to provide evidence that Early Endoscopic intervention benefits patients with acute gallstone pancreatitis and biliopancreatic obstruction.
  - The persistence of main bile duct stones does not by itself contribute to worsening or persisting pancreatic inflammation. If acute cholangitis can be safely excluded, EEI is not mandatory and should not be considered a standard indication.
- **ERCP on reduction of complications**
  - odds of having complications are reduced in predicted severe disease by Early ERCP but renders non-significant effect in predicted mild disease and for reduction of mortality in either predicted or severe disease. (Khurram et. Al, 2009)



# References

- Ayub, K. et. Al., Endoscopic retrograde cholangiopancreatography in gallstone-associated acute pancreatitis, Cochrane upper Gastrointestinal and Pancreatic Diseases group, Oct 2009.
- Fauci, et.al. Harrison's Principles of Internal Medicine. (2008). USA: Mc-Graw Hill Companies Inc. 17<sup>th</sup> edition.
- Oria, A. , Early Endoscopic Intervention Versus Early Conservative Management in Patients With Acute Gallstone Pancreatitis and Biliopancreatic Obstruction A *Randomized Clinical Trial, Annals of Surgery, 2007; 245- 1: 10-17*
- Sleisenger & Fordtrans's GI and Liver Disease (2003). Missouri: W.B. Saunders Company.
- Bickley, L. Bates' Guide to Physical Examination and History Taking. (2007). Lippincott Williams & Wilkins. 9<sup>th</sup> edition.
- 
- 

