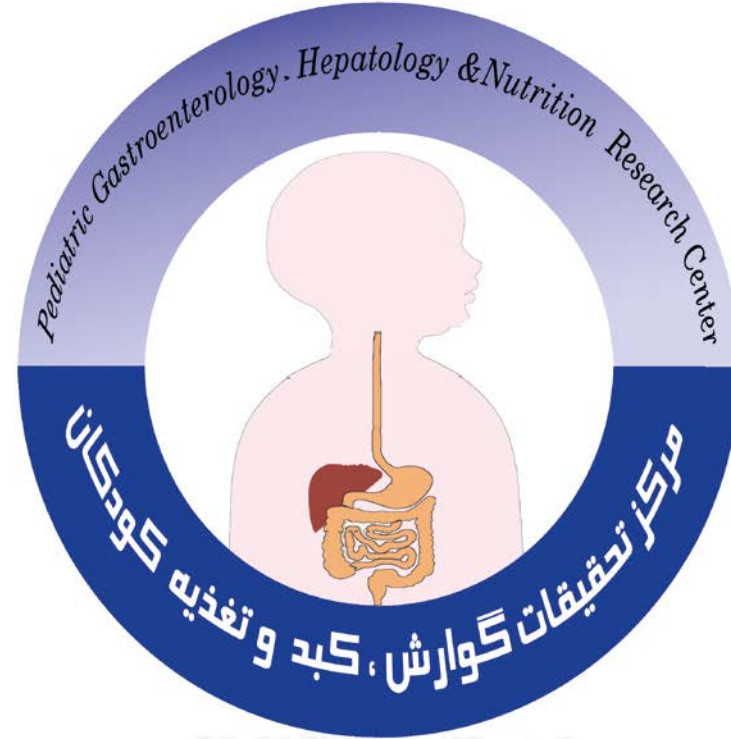




Pancreatitis

Dr. Shiva Mohammadi

Fellowship of Pediatric Gastroenterology



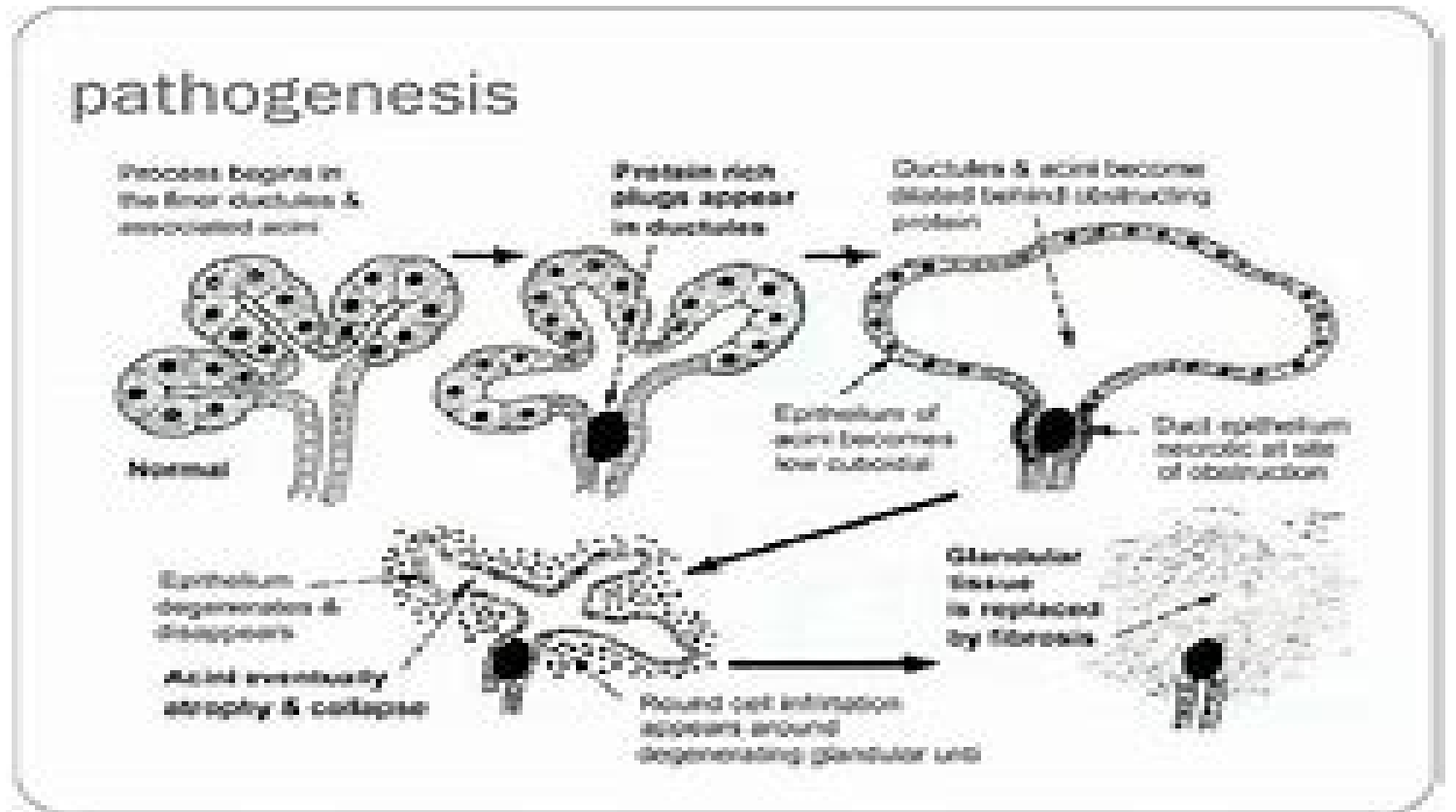
Mofid Children's Hospital
Shahid Beheshti University of Medical Sciences

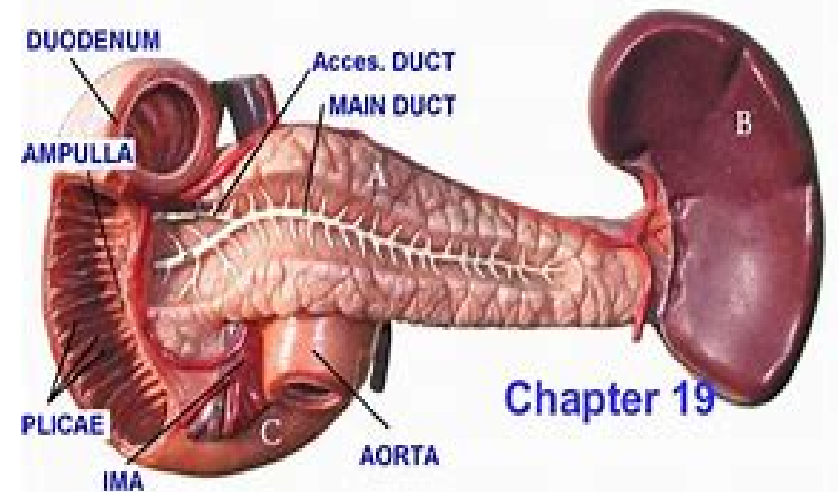
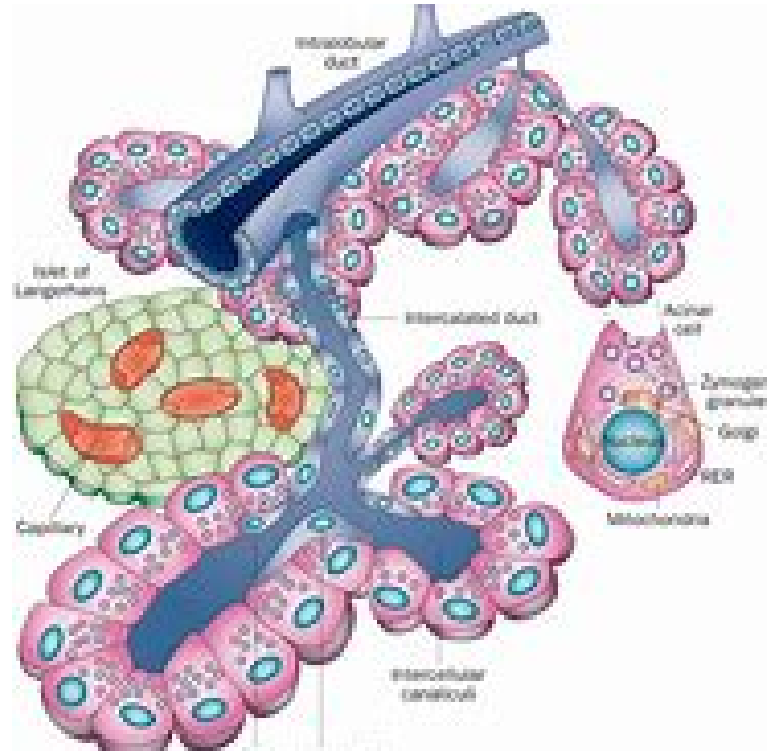
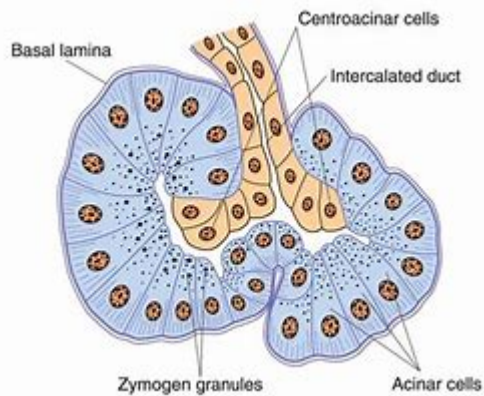
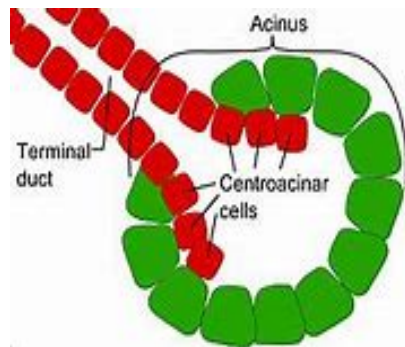


Dr. Shiva Mohamadi
Subspeciality Of Gastroenterology And Hepatology
Mofid Hospital

Acute Pancreatitis

- Clinically = A **sudden onset of abdominal pain** + **↑ Digestive enzymes in the blood or urine.**
- Mechanism = Premature activation of trypsinogen.
- (Trypsinogen Activation Peptide or TAP is one of the earliest markers)





- ***ERCP associated pancreatitis*** can be attenuated by pretreatment with trypsin inhibitors (gabexate)
- **Mutations** that enhance trypsinogen activation , diminish trypsin inactivation , or limit trypsin clearance from the ducts are associated with acute pancreatitis.
- Secondly a **vigorous immune response** contributes to the **severity** of the pathological condition

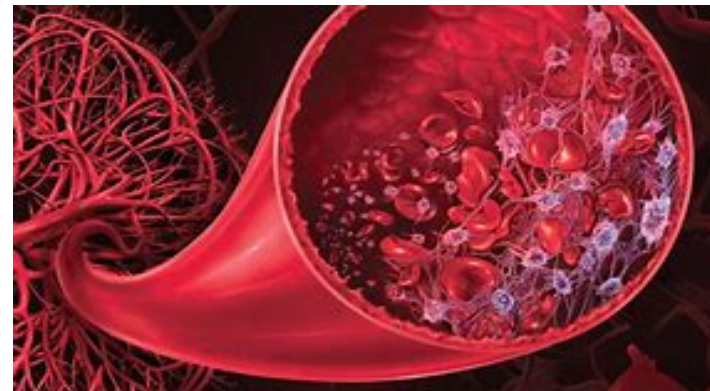
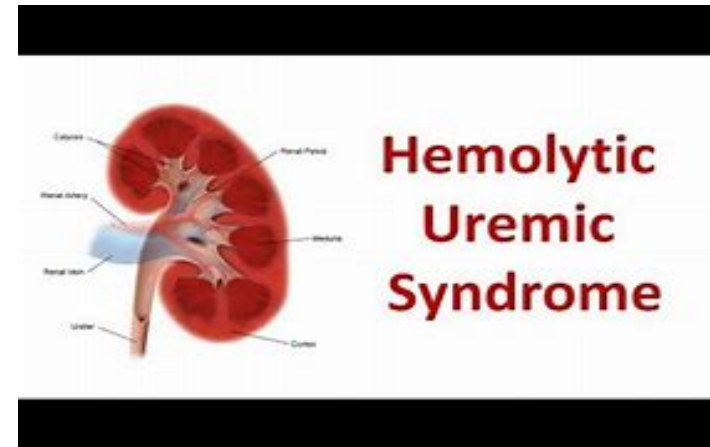


Etiology Of Acute Pancreatitis In adults:

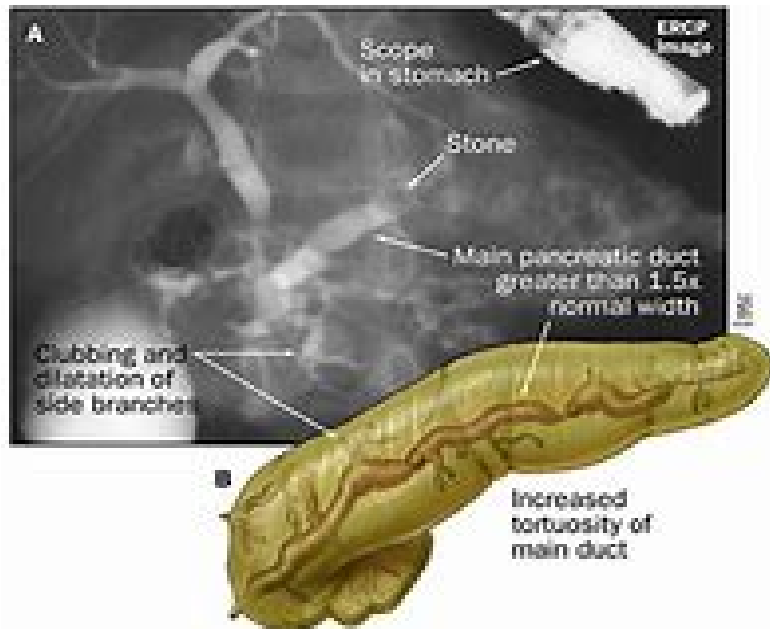
- **Gallstones**
- **Alcohol abuse**
- **Hypercalcemia**
- **Hypertriglyceridemia**
- **Medications**
- **Blunt trauma**

In Children:

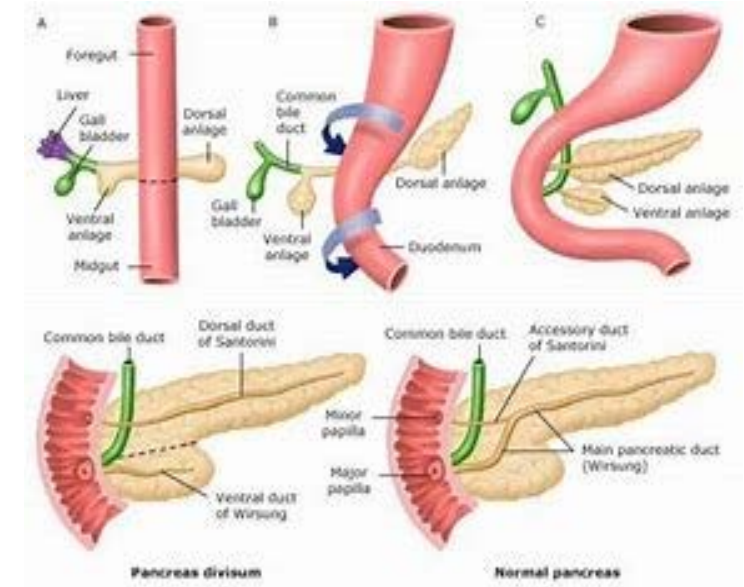
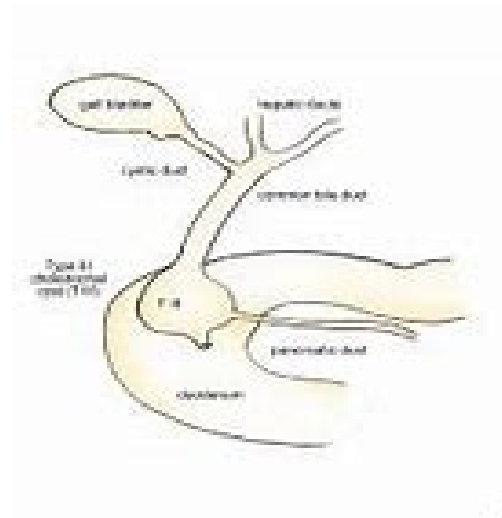
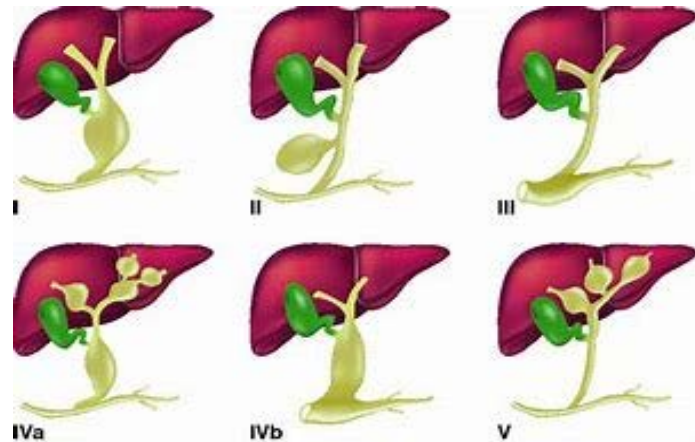
- **Severe systemic illnesses** (20% of reported cases)
 - * Death due to acute pancreatitis in children alone are now uncommon (<2%)
 - * **HUS** = The **most common** of all systemic disease causing acute pancreatitis
 - * A.P after organ transplantation is also common.



- **Gallstones/Sludge** = An important cause of A.P in children = In 10-30% = Therapeutic ERCP



Structural abnormalities = The most common : **Pancreas divisum**.
 choledochal cysts, choledochoceles, partial pancreas divisum.



- **Infectious** acute pancreatitis (primarily viral) includes:
- *Mumps, Entrovirus, EBV, HAV, CMV, Rubella, Coxsackie virus, Varicella, Rubeola, measles and influenza virus.*
- *HIV* = Frequently develop A.P usually from a secondary infection (CMV, MAI, PCC, Cryptosporidium parvum,)/from medications.
- Bacterial infections
- Helminth infections(Ascaris lumbricoides) = Can be severe, complicated, and difficult to treat.



Medications

- **The strongest evidence in adults:**
- **Azathioprine**
- **6MP**
- **Thiazide diuretics**
- **Sulfonamides**
- **Furosemide**
- **Estrogens**
- **Tetracycline**
- **Suggestive evidence for:**
- **L-asparaginase**



Mofid Children's Hospital
Shahid Beheshti University of Medical Sciences

- **Iatrogenic hypocalcemia**
- **Chlorthalidone**
- **CS**
- **Ethacrynic acid**
- **Phenformin**
- **Procainamide**



- **25% Of ped.cases were associated with the use of medications.**
- **Of this patients , 65% had no other etiology.**
-
- **Mezalamine , CTX in the most recent series**
- **Anticonvulsant valproate = The most common medication reported in this series**
- **The medication age was 8.9 years(range 1-18years)**
- **Min dose: 4.5 mg/kg/d(range 20-85)**

- ▶ **Trauma** , blunt/accidental/child abuse (**common cause = 20%** of cases)
- ▶ The major concern = pancreatic duct transection
- ▶ **Post-ERCP** pancreatitis
- ▶ **Familial** pancreatitis includes hereditary
- ▶ **Atypical CF** with compound heterozygous CFTR mild-variable mutations or CFTR mutations plus modifying factors , such as **SPINK1** mutations , are the most common cause of familial pancreatitis in our experience.
- ▶ There is no association between α 1-antitrypsin mutations and acute pancreatitis



Evaluation of a child with Acute Pancreatitis should include:

- **Ca – TG – R/O of other metabolic disorders – DKA and inborn errors of metabolism.**

Acute Recurrent Pancreatitis (ARP)

- Is seen in **10%** of children after a 1st episode of acute pancreatitis
- **25%** of cases are idiopathic.
- **Congenital structural anomalies** of the biliary tree , pancreas and intestinal tract
- **Genetic mutations**
- A careful evaluation aimed at identifying reversible causes should be undertaken
- In addition to avoiding another attack of acute pancreatitis one should consider the prevention of chronic pancreatitis through eliminating known risk factors.
- Theoretically the progression to chronic pancreatitis may be slowed by the use of **antioxydants**
- (By reducing the frequency and severity of recurrent symptoms in hereditary pancreatitis) but this approach is currently unproven.

Diagnosis of Acute Pancreatitis

- **Sudden onset** of typical **abdominal pain** + Elevation of **amylase or lipase** to at least **3 times the ULN**
- But both enzymes can be normal when there is radiographic and clinical evidence of pancreatitis.
- Also both enzymes can be elevated by other conditions unrelated to pancreatitis.
- The **level** of elevation is also **not diagnostic** , **although** the higher the level is above the URL the **more likely** there is to be pancreatic inflammation
- Other pancreatic products: **PL A2 , Trypsin , TAP , Elastase** are elevated in pancreatitis.
- Elevation of AST and ALT. **↑[AST- ALT] + [amylase or lipase]** may be more predictive of pancreatitis than **↑amylase or lipase** alone.
- Paine is usually **epigastric , RUQ or LUQ** with **radiation through to the back.**



- But back pain can occur alone or may localize to other areas of the abdomen.** •
- Nausea/Vomiting** •
 - Fever** •
 - Tachycardia** •
 - Hypotension** •

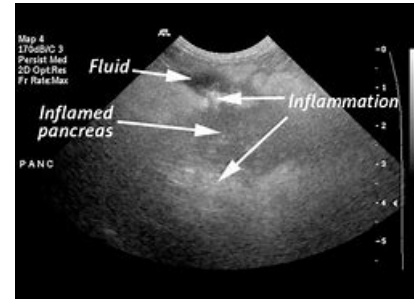
- **Jaundice**
- **Guarding , rebound , ↓bowel sounds**
- **Feeding intolerance**
- **Children < 3yr : Atypical symptoms = irritability , abdominal distention and fever**
- **Ascites or abdominal mass**
- **Epigastric tenderness (but nonspecific and unreliable)**

Pancreatic Edema > Necrosis > Hemorrhage

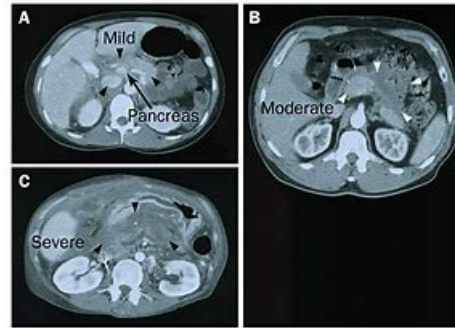
Mild Acute Pancreatitis	Severe Acute Pancreatitis
<ol style="list-style-type: none"> 1. Severe abdominal pain in epigastric area /RUQ 2. Vomiting 3. Fevere 4. Abdominal distention 5. Abdominal tenderness 6. +/- Abdominal mass 7. +/- Dehydration <p>Prognosis: Good (If no complication)</p> <p>Lab data:</p> <ol style="list-style-type: none"> 1. High Lipase(<u>choice and more specific</u>) 2. High Amylase 	<p>Patient is ill/toxic/with severe abdominal pain/nausea/vomiting/high grade fevere/jaundice/ascites/pleural eff.(in <u>left</u> side)/shock/gray Turner and Cullen sign/necrotic pancreas(a hemorrhagic inflammatory mass)</p> <p>Prognosis: bad</p> <p>Lab data: High : Hct-WBC-BS-Urine Glu-GGT-PT/INR-Bil <u>Low : Ca</u></p> <p>❖ CT is recommended for determination of severity</p>



US



CT scan





Medical Management of Acute Pancreatitis



- **Analgesia**
- **IV fluids**
- **Pancreatic rest**
- **Monitoring for complications**
- **Volume expansion early the course of acute pancreatitis is important for :**
 - ***Cardiovascular stability**
 - ***Preventing development of pancreatic necrosis**

Full attention to fluid balances because:

1. Patients kept without food

2. Third spacing

3. Fluid loss from NGT used to decompress the stomach(as a Tx for vomiting)



- **Meperidine** 1-2 mg/kg IM or IV for pain control
- **Enteral/Total parenteral nutrition** are often unnecessary in mild cases but should be instituted early if a severe or prolonged course is anticipated.
- **Antibiotics** are usually unnecessary except for the most severe cases especially if significant necrosis is present.



Severity of Acute Pancreatitis

- Acute pancreatitis can be life-threatening
- The early causes of death:
- **1. cardiovascular collapse** = vascular leakage syndrome with 3rd spacing of fluid + vomiting + making the patient NPO.
- Tx: Fluid resuscitation guided , by following CVP.
- **2. respiratory failure = ARDS like** (leakage of fluid into alveolar spaces + inflammation)

- **Late life-threatening complications :**
- **are related to infected pancreatic necrosis and multiple organ failure (pancreatic necrosis is uncommon in children : 0.3%)**
- **How we can help limit these late complications ?**
- **1.Judicious use of antibiotics**
- **2.Attention to nutrition**



Complications of Acute Pancreatitis

- **Local :**
- Edema
- Inflammation
- Fat necrosis
- Phlegmon
- Pancreatic necrosis
- 1.Steril
- 2.Infected
- Abscess
- Hemorrhage
- Fluid collections
- Psuedocysts
- Duct rupture and strictures
- Extention to nearby organs



Mofid Children's Hospital
Shahid Beheshti University of Medical Sciences

- **Systemic :**
- Shock
- Pulmonary edema
- Pleural effusion
- Acute renal failure
- Coagulopathy
- Hemoconcentration
- Bacteremia/sepsis
- Distant fat necrosis
- Vascular leak syndrome
- Multiorgan system failure
- Hypermetabolic state
- Hyperglycemia



Pathophysiology of pancreatitis

- **Distruption/obstruction of ducts > Change trypsinogen into trypsin > activation of other proenzymes > Autolysis + Pancreastasis**
- **Edema > Necrosis (localased or diffused) > Rupture of vessles > Hemorrhage > Inflammatory response in peritoneum**



Diagnostic criteria

2 of 3 :

1) Abdominal pain

2) Amylase or Lipase ≥ 3 [ULN]

3) Imaging findings

Cullen / Gray Turner

