
بسم الله الرحمن الرحيم

Acute Pancreatitis
Diagnosis & Management

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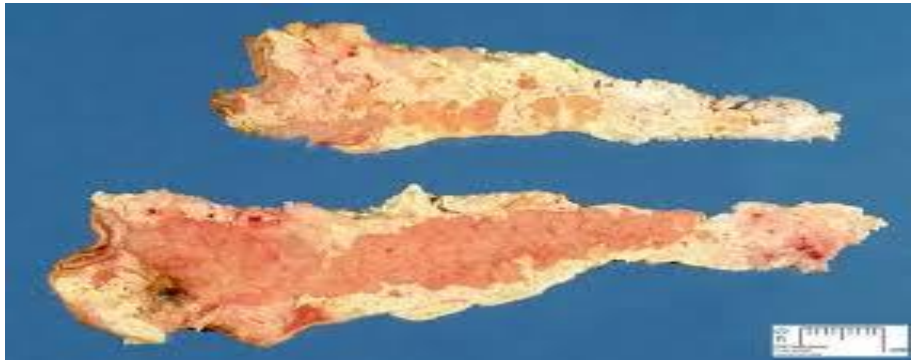
Introduction

- Acute pancreatitis is an acute inflammatory process of the pancreas
- Overall mortality rates in acute pancreatitis are approximately 2%
- but can be as high as 30 % in patients with persistent organ failure (severe acute pancreatitis)



UptoDate

- Estimates of incidence : 3.6 - 13.2 cases per 100,000 children annually.

- Children may present with only a single episode;
  however, up to 30% of patients progress to acute recurrent pancreatitis and/or chronic pancreatitis.

- Acute pancreatitis is a reversible inflammatory process with changes
- to the pancreatic parenchyma and/or function without lasting effects.

- Chronic pancreatitis is an irreversible process with changes to parenchyma and/or function of the pancreas.

Acute pancreatitis classification : (based on NASPGHAN)

- Mild: no local or systemic complications or exacerbation of existing comorbid disease

- Moderately severe, includes presentations with local or systemic complications, exacerbation of an existing comorbid disease, or transient (<48 hours) organ dysfunction

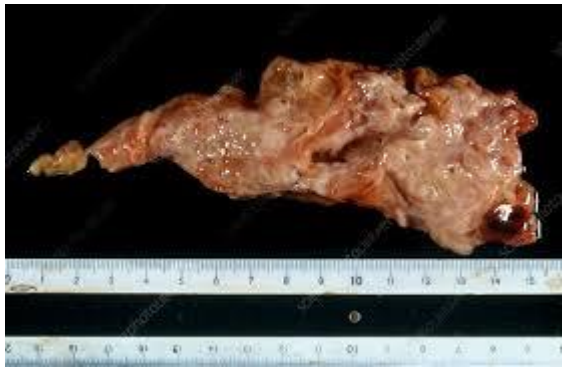
- Severe acute pancreatitis. includes organ dysfunction persisting greater than 48 hours.

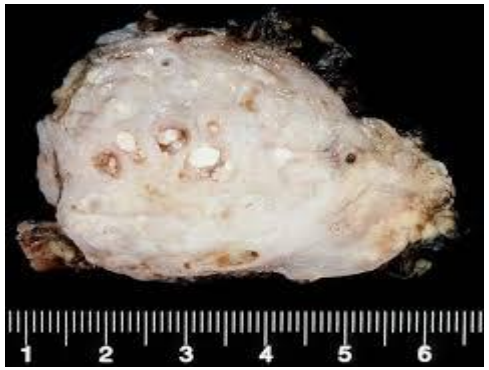
- Acute recurrent pancreatitis is defined as greater than one episode of acute pancreatitis with either:

(1) a 1-month pain-free interval between diagnoses or

(2) normalization of amylase and lipase and resolution of pain before a subsequent episode of pancreatitis is diagnosed.

Chronic pancreatitis





Criteria

1- Abdominal pain consistent with pancreatic origin and radiographic findings suggestive of chronic pancreatic damage

2- Evidence of exocrine pancreatic insufficiency and suggestive pancreatic radiographic findings,
or

3- Evidence of endocrine pancreatic insufficiency and suggestive pancreatic imaging findings.

Chronic pancreatitis may also be

- diagnosed based on pancreatic biopsy or surgical specimen with compatible histopathologic features.

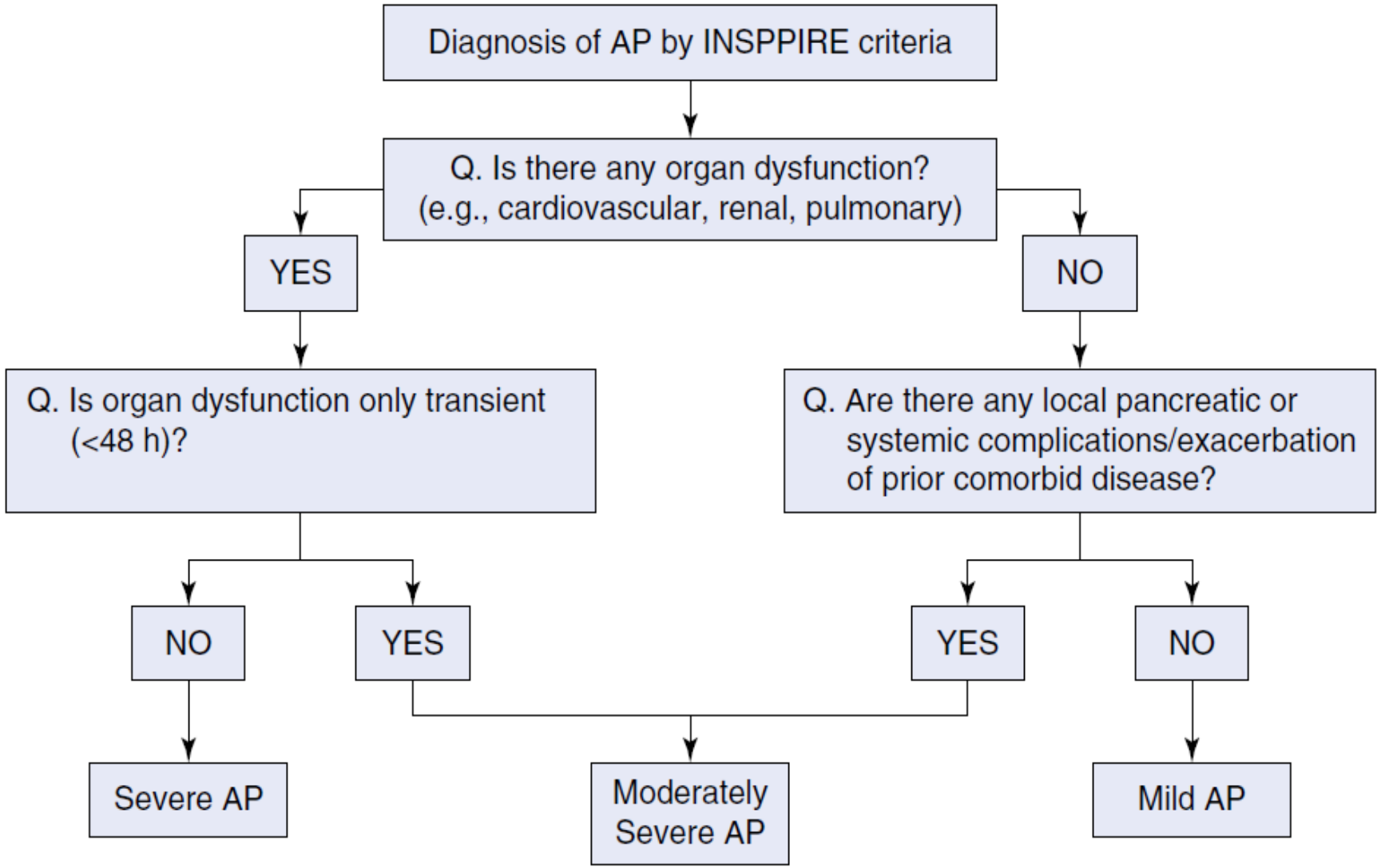


Fig. 82.1 Classification of Acute Pancreatitis.[16] *AP*, Acute pancreatitis. *INSPPIRE*, INternational Study group of Pediatric Pancreatitis: In search for a cuRE. (Printed with permission from Wolters Kluwer Health, Inc.)

Etiology

Opposed to in adults, where gallstone and alcohol-induced pancreatitis are common, the etiologies of acute pancreatitis in children are variable, including

- Biliary or obstructive factors,
- Systemic disease 10 – 50 %
- Medication induced
- Trauma
- Metabolic disease
- Idiopathic

Clinical Manifestations

TABLE 82.1 Signs and Symptoms of Acute Pancreatitis

	Common	Uncommon
Symptoms	Abdominal pain	Back pain
	Irritability (infants)	Jaundice
	Nausea	Fever
	Vomiting	Feeding intolerance
	Anorexia	Respiratory distress
Signs	Abdominal tenderness	Grey Turner sign
	Abdominal distension	Cullen sign
	Evidence of dehydration	Evidence of ascites
		Evidence of pleural effusion

Diagnosis

The diagnosis of acute pancreatitis in a child can be made by fulfilling two of three accepted criteria:

- abdominal pain compatible with pancreatic origin,
- amylase and/or lipase at least three times the upper limit of normal,
- radiographic evidence of pancreatitis

Laboratory Studies

- Serum amylase and lipase levels are measured to screen for pancreatitis.
- Lipase has a higher sensitivity than amylase

- Some patients may have clinical and radiologic evidence of acute pancreatitis without elevated amylase and/or lipase

- Serum lipase usually rises 3 to 6 hours after onset of acute pancreatitis, with a peak at 24 hours, and remains elevated for up to 8 to 14 days.

- Amylase also rises within 3 to 6 hours of onset and may be elevated for up to 5 days but can normalize in 24 hours.

- Infants present a challenge because amylase and lipase expression are low at birth and increase to adult range through the first year of life—careful interpretation of values in infants

- In any patient with suspected acute pancreatitis, both amylase and lipase should be measured initially.

- In the absence of a known etiology measuring
  aminotransferases,
  conjugated and unconjugated bilirubin,
  gamma-glutamyl transferase (GGT),
  triglycerides, serum glucose
  calcium.

Amylase and lipase are not necessarily specific for acute pancreatitis;

other conditions causing elevated amylase and lipase cause mild elevations

< 3 xULN :

- renal failure
- liver damage
- intestinal inflammation
- diabetic ketoacidosis
- head trauma.
- macroenzymemia may be > 3xULN
- Familial or sporadic amylase or lipase Levels greater than three times the upper limit of normal without evidence of pancreatitis macroenzymes.

Imaging

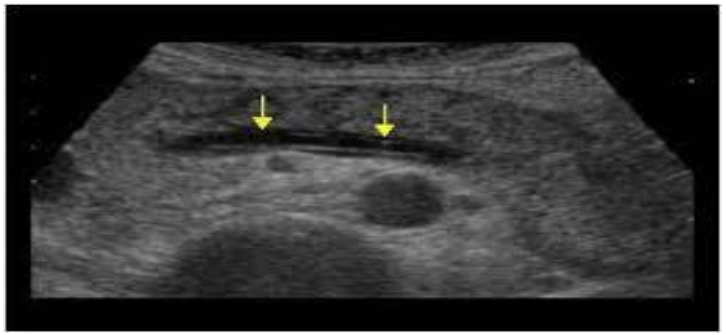
- Transabdominal ultrasound (TUS)
- Computed tomography (CT)
- Magnetic resonance imaging (MRI) or MRCP

Imaging may not necessary in patients who already meet criteria for diagnosis of acute pancreatitis, unless:

- suspicion for moderately severe or severe pancreatitis
- pancreatitis of biliary origin
- typical pancreatic pain and minimally elevated enzymes

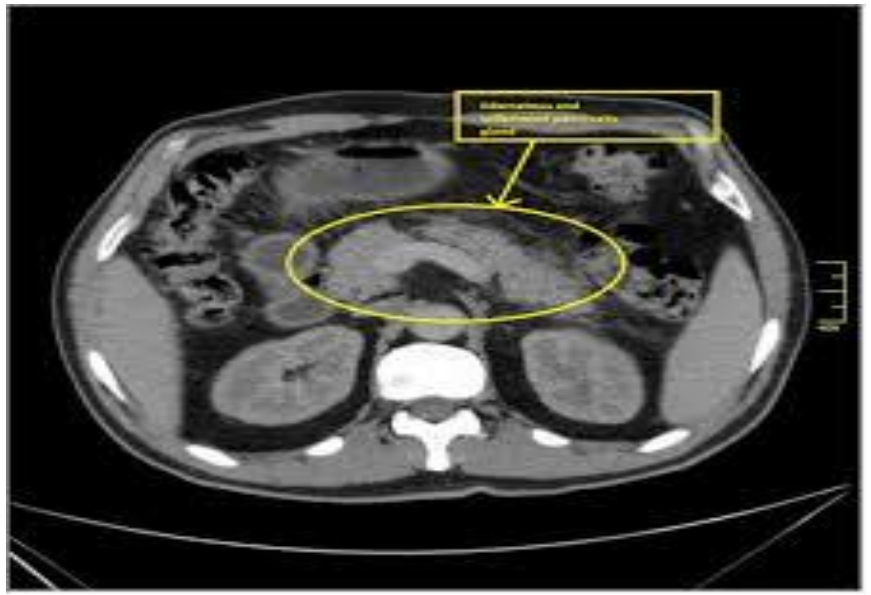
- TUS is a good initial choice for imaging , the pancreas is generally well visualized, along with the bile duct and gallbladder, helpful in the assessment for biliary related pancreatitis (e.g., acute pancreatitis with elevated bilirubin or serum transaminase levels).

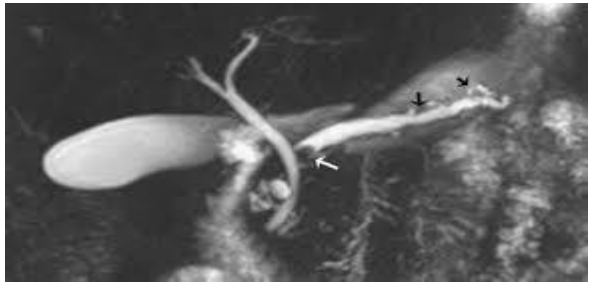
- Although TUS does remain the initial test of choice, it is important to realize limitations in clinical interpretation.



- CT with IV contrast provides a more comprehensive view of the pancreas and its surrounding structures, along with the remainder of the abdomen good sensitivity to identify calcific calculi within the pancreatic ducts or calcific deposits within the pancreatic parenchyma.

One disadvantage is that CT imaging does not provide good visualization of ductal anatomy.

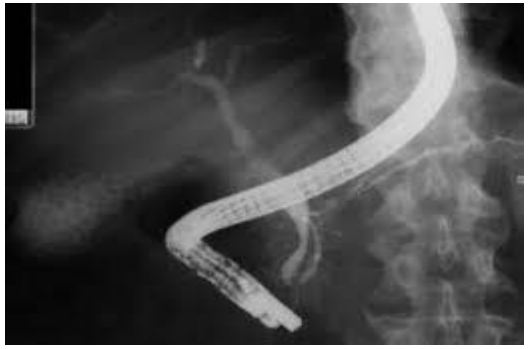




- MRI or MRCP : assess pancreatic disease or its complications.
- It identifies parenchymal abnormalities , visualization of the pancreas ducts & biliary ductal system and gallstones , pancreas divisum , choledochal cysts, or anomalous pancreaticobiliary junction.

- Secretin can be administered to stimulate pancreatic bicarbonate secretion to enhance views of the pancreatic ductal system.

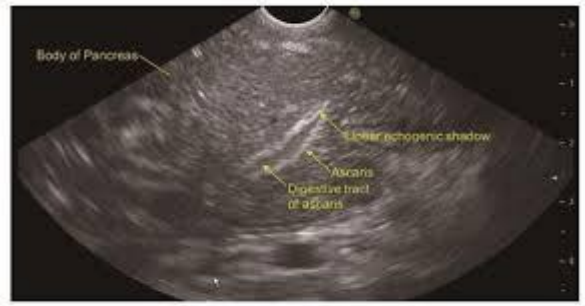
- Disadvantages of MRI : scan duration of up to an hour
  lower sensitivity for pancreatic calcification identification.
  limitation of anesthesia for younger children



- ERCP and endoscopic ultrasound (EUS), also have roles in imaging the pancreas.

- ERCP should be used mainly as a therapeutic measure when necessary

- EUS is safe in children and in adults it offers a higher sensitivity for detection of gallstones or microlithiasis than less-invasive imaging modalities.
  - more detailed view of the pancreas and surrounding strictures,
  - capacity for therapeutic applications.



- After a patient's first episode of acute pancreatitis, an extensive work-up is generally not necessary, because 70% or less of these patients will not have another episode of pancreatitis.

- All patients with a first episode should have serum calcium and triglycerides levels measured

- If there is family history of pancreatitis, genetic testing should be considered.
- Genetic testing should be strongly considered in patients with greater than one episode of acute pancreatitis.

- Jaundice should prompt further work-up with imaging investigating for biliary pancreatitis.

- In cases of trauma, CT and MRCP should initially be considered to investigate for pancreatic duct disruption.

Management

- Supportive care.
- The mainstays of treatment include intravenous fluid hydration,
- Pain control
- Nutrition management
- Clinical monitoring for complications.
- Endoscopic and surgical management in selected complicated cases. .

Indications for ICU admission

- Severe acute pancreatitis
- Acute pancreatitis and one or more of the following parameters:

  Pulse <40 or >150 beats/minute

  Systolic arterial pressure <80 mmHg or mean arterial pressure <60 mmHg

  or diastolic arterial pressure >120 mmHg

  RR >35 breaths/minute

  Serum Na <110 mmol/L or >170 mmol/L

  Serum K <2.0 mmol/L or >7.0 mmol/L

  PaO2 <50 mmHg

  pH <7.1 or >7.7

Cont,

Serum glucose >800 mg/dL

Serum Ca >15 mg/dL

Anuria

Coma

persistent (>48 hours) SIRS

Elevated Hct (>44 percent), BUN 20 mg/dL, or Cr >1.8 mg/dL

Age >60 years

Underlying cardiac or pulmonary disease, obesity

Fluids

Early aggressive fluid resuscitation associated with better outcomes in the pediatric population, including cardiovascular stability and preventing development of pancreatic necrosis

Aggressive intravenous fluid is usually considered to be a rate of 1.5 to 2 times maintenance fluid during the first 24 hours

Children who have any evidence of hemodynamic compromise at admission should be administered a bolus of 10 to 20 mL/kg

The optimal type of intravenous fluid for resuscitation has been a topic of debate

Isotonic crystalloid fluid should be used, lactated Ringer solution.

- But use of normal saline , half normal saline with dextrose, or lactated Ringer solution is acceptable

Monitoring of Children Diagnosed With Extra-Pancreatic Manifestations of Acute pancreatitis

- Multiorgan disease in adult patients with AP , over half of AP-related deaths occurring within 1 week of the onset of multiorgan dysfunction

- Cardiac, pulmonary and renal involvement

- key components of adult scoring systems used to predict severity of an AP episode including the modified Atlanta Classification, Ranson criteria, Japanese Severity Assessment, Glasgow Score, BISAP, and APACHE II ,

  But, not validated in pediatrics

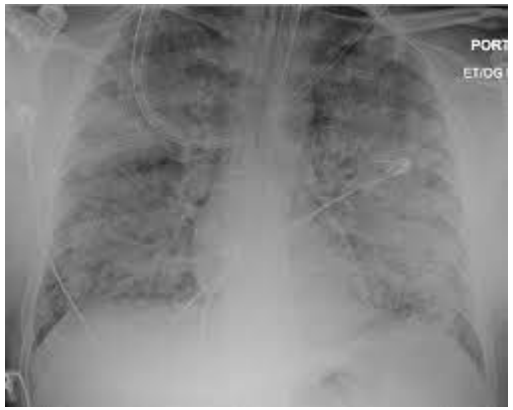
Cardiovascular Monitoring

- Hypovolemia - strong predictor of morbidity and mortality among adults and correlated with the magnitude of the SIRS

- Tachycardia utilized for severity of AP, its improvement confirm adequate fluid resuscitation in addition to monitoring urine output and skin turgor

- Routine vitals every 4 hours
- Rare cases of cardiac tamponade and atrial fibrillation (unexplained hypotension, shortness of breath and/or chest pain)

Pulmonary Monitoring



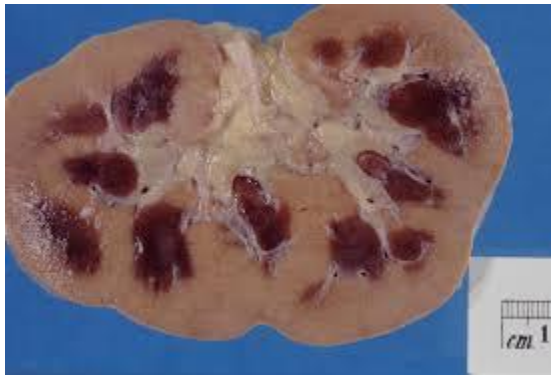
- Early complications (within the first 48 hours): ARDS syndrome (most common)
  pneumonia,
  pulmonary edema/effusions

1- Monitor O2 sat

2- some advocating patient beds be elevated at 30-degree angle to decrease likelihood of pulmonary sequestration

Renal Monitoring

- Acute kidney injury (AKI) via abdominal compartment syndrome
  or inflammatory-driven damage to the proximal tubule



- Marked by elevation of BUN and creatinine (esp. in the first 48hrs)
  with decreased urine output,

- BUN alone in some adult AKI associated with a 10-fold increased risk of mortality in severe AP

- In rare cases, continuous veno-venous hemofiltration is required to prevent further kidney damage, prevent abdominal compartment syndrome, and/or removae inflammatory cytokines,

Nutrition

- Although in the past NPOinitial therapy for patients with acute pancreatitis, substantial pediatric evidence currently supporting early enteral nutrition in children with mild acute pancreatitis. at or within the first 24 hours of admission

- adult literature with strong support for enteral nutrition, shown to increase survival and reduce complications except direct contraindications to enteral feeds

- If tube feeding is required, nasojejunal feeding, is preferred but NG feeding is also possible even in severe acute pancreatitis.

- If inability to tolerate enteral nutrition due to persistent pain, ileus, or other complication, parenteral nutrition should be considered if there is no ability for enteral intake for 5 to 7 days.

- There are no clear guidelines or evidence for or against initial enteral nutrition in acute pancreatitis associated with pancreatic ductal disruption.

- Finally, there is no evidence for improved recovery in low fat diet as compared with a standard fat diet.

- Adult studies support enteral nutrition in severe acute pancreatitis with lower rates of multiorgan failure, shorter hospital stays, and lower mortality rates.

Pain Relief

- Acetaminophen
- Meperidine 1-2 mg/kg IM or IV
- with increase to parenteral opioid medication (morphine or other) if pain is inadequately controlled.
- If available, specialized pain management services .

Complications

Local or systemic complications include :

- Peripancreatic fluid collections

  pseudocysts

  acute necrotic fluid collection

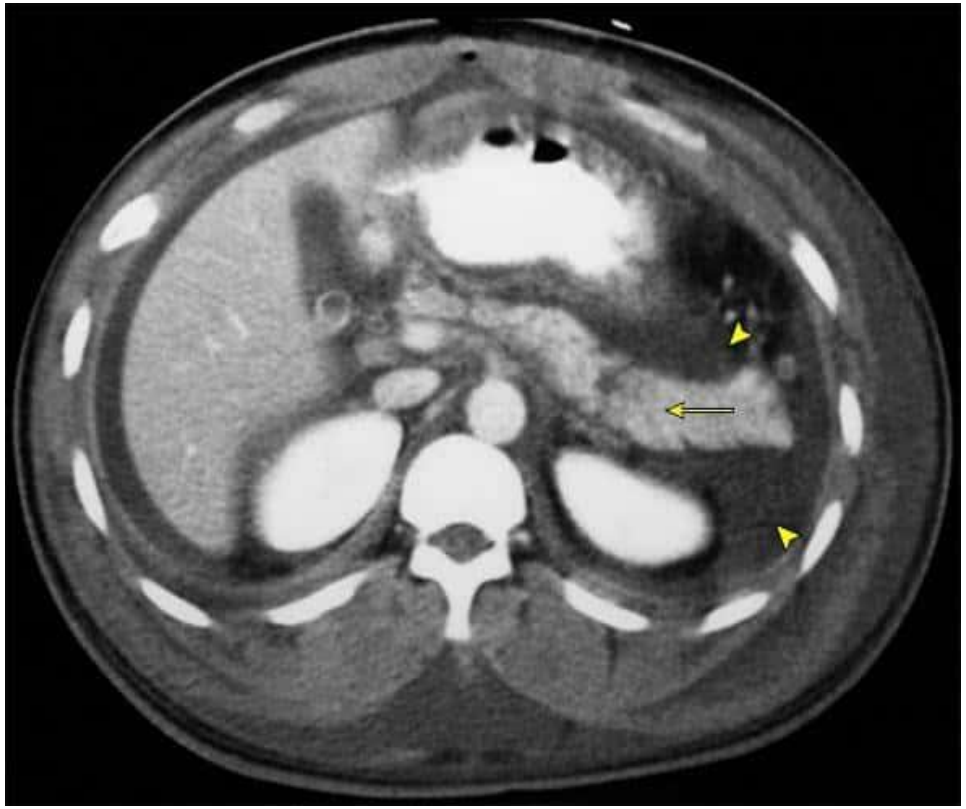
  walled-off necrosis.

Organ dysfunction

- Includes respiratory, Renal, or cardiovascular dysfunction

Acute peripancreatic fluid collection(APFC)

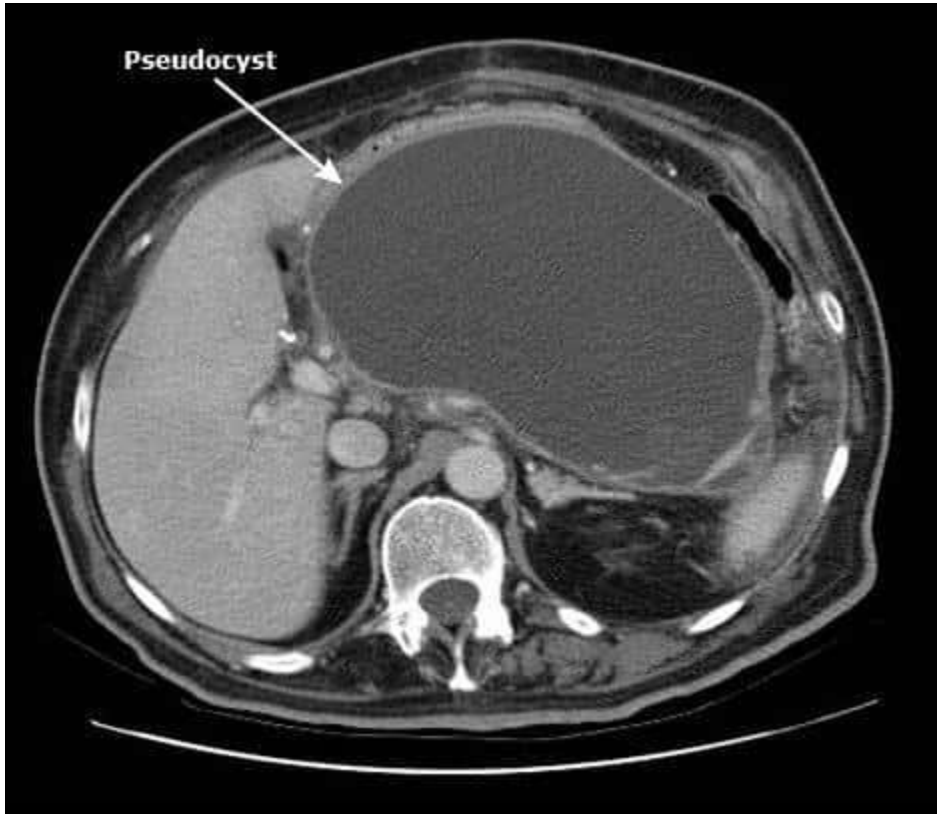
- Fluid collections usually develop in the early phase of pancreatitis.
- Do not have a well-defined wall
- Usually remain asymptomatic
- Usually resolve spontaneously without the need for drainage
- Mostly resolved within 7 to 10 days, with only 6.8 percent of APFCs persisting beyond four weeks as pancreatic pseudocysts



Pancreatic pseudocyst

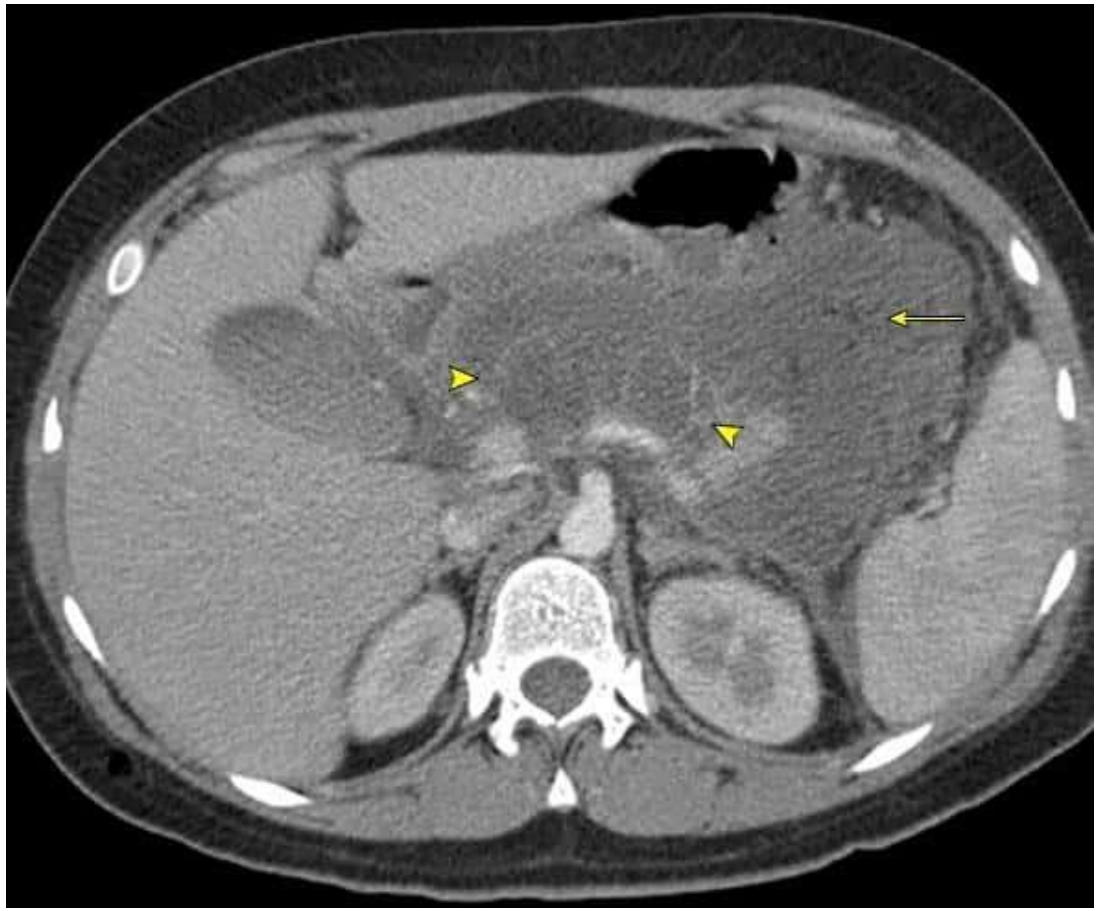
- Encapsulated collection of fluid with well-defined inflammatory wall usually outside pancreas with minimal or no necrosis

- Usually occur more than four weeks after onset of interstitial edematous pancreatitis.



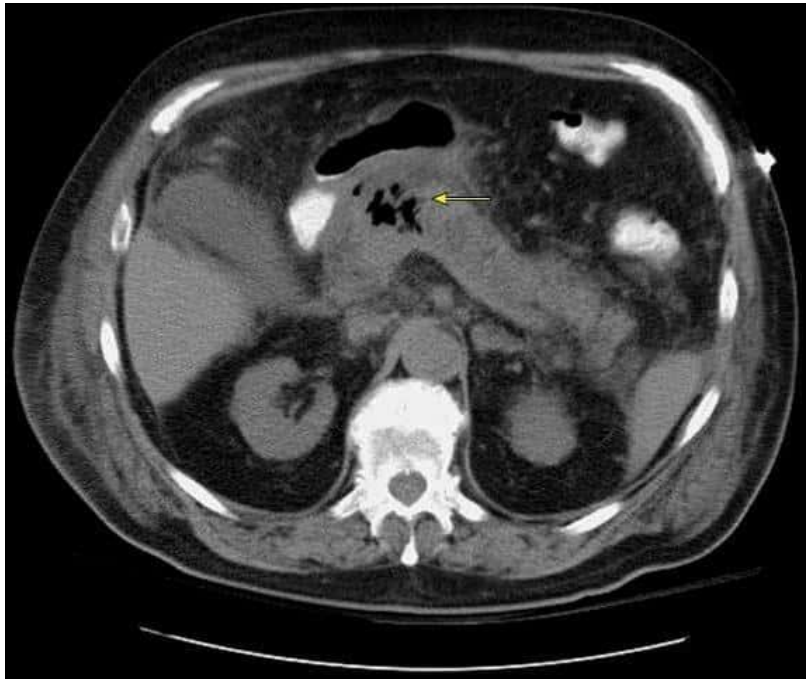
Acute necrotic collection and walled-off necrosis

- Necrotizing pancreatitis most commonly manifests as necrosis involving both the pancreas and peripancreatic tissues.
- Necrosis may result in an acute necrotic collection (ANC) that contains a variable amount of fluid and necrosis but lacks a definable wall (image 3)
- or walled-off necrosis (WON), which consists of a mature, encapsulated collection of pancreatic and/or peripancreatic necrosis that has developed a well-defined inflammatory wall.
- Both ANC and WON are initially sterile but may become infected



Infected necrosis

- one-third of patients with pancreatic necrosis develop infected necrosis
- It can occur early course of necrotizing pancreatitis, it is more often seen late in the clinical course (after 10 days)
- The majority of infections (approximately 75%) are monomicrobial with gut-derived organism s (eg, E. coli, Pseudomonas, Klebsiella, and Enterococcus).
- However, the incidence of gram -positive, multidrug resistant and fungal organisms in infected pancreatic necrosis is rising.
- It should be suspected in patients with pancreatic or extrapancreatic necrosis who deteriorate (clinical instability or sepsis physiology, increasing white blood cell count, fevers) or fail to improve after 7 to 10 days of hospitalization.



- Clinical signs of infection and abdominal imaging with the presence of gas within the necrosis suggestive of infection and antibiotic therapy can be initiated without aspiration

- Empiric antibiotics known to penetrate pancreatic necrosis (eg, a carbapenem alone; or a quinolone, ceftazidime, or cefepime combined with an anaerobic agent such as metronidazole)

- fail to improve in 48 - 72 hrs or clinical deterioration, we perform drainage or debridement of pancreatic necrosis (necrosectomy) or CT-guided aspiration

- In stable patients with infected necrosis, delay drainage/necrosectomy by continuing antibiotics for at least four weeks

Sterile necrosis

- If the aspirated material on CT-guided FNA is sterile, we discontinue antibiotics and continue conservative treatment for four to six weeks.

- Use of antibiotics is not recommended. Sterile necrosis does not require therapy.

- Indications for intervention (radiological, endoscopic, or other minimally invasive) if no signs of infection (eg, fever, hypotension, leukocytosis) include:

1- Ongoing gastric outlet, intestinal, or biliary obstruction due to mass effect at least four weeks

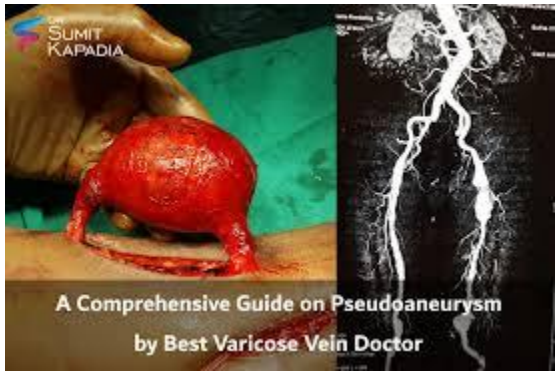
2- Persistent symptoms (eg, abdominal pain, nausea, vomiting, anorexia or weight loss) >8 weeks

3- Disconnected duct syndrome (full transection of the pancreatic duct) with persisting symptomatic collections with necrosis (eg , pain, obstruction) >4 weeks

Peripancreatic vascular complications

- Splanchnic venous thrombosis — (splenic, portal, and/or superior mesenteric veins) 1 to 24%

- Pseudoaneurysm — rare but serious complication ,
  should be suspected when unexplained gastrointestinal bleeding
  unexplained drop in hematocrit,
  or sudden expansion of a pancreatic fluid collection.



Abdominal compartment syndrome — defined as sustained intra-abdominal pressure >20 mmHg
due to tissue edema from aggressive fluid resuscitation, peripancreatic inflammation, ascites, and ileus

Systemic complications

- Increased risk for exacerbation of underlying comorbidities

  coronary artery disease

  chronic lung disease

  prediabetes & diabetes

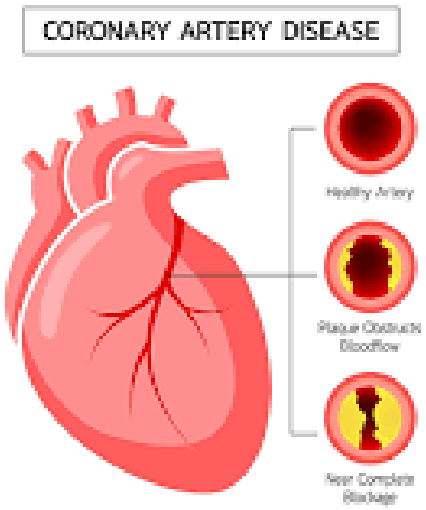


TABLE 82.2 Complications of Acute Pancreatitis in Children

Local	Systemic
Inflammation	Shock/vascular leak syndrome
Edema	Pulmonary edema
Pancreatic necrosis	Pleural effusions
Fat necrosis	Coagulopathy
Fluid collections/pseudocyst	Acute renal failure
Phlegmon	Dehydration
Abscess	Sepsis
Hemorrhage	Distant fat necrosis
Pancreatic duct rupture	Multiorgan system failure
Extension to nearby organs and vessels	Hypocalcemia
	Hyperglycemia

References :

- Management of Acute Pancreatitis in the Pediatric Population: A Clinical Report From the North American Society for Pediatric Gastroenterology, Hepatology and Nutrition Pancreas Committee 2018
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