Pancreatitis

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Introduction

- Acute pancreatitis: sudden inflammation of the pancreas.
- Triggered by premature activation of pancreatic enzymes.

PATHOPHYSIOLOGY

ACUTE PANCREATITIS

- Acinar cell injury :destruction of pancreatic and peripancreatic tissue
- The gland may be swollen, edematous, hemorrhagic, or even necrotic
- Histology :interstitial edema and inflammation to hemorrhage and necrosis
- The disease ranges in severity from mild and self-limited to severe and life threatening
- The Atlanta Classification: acute pancreatitis as an acute inflammatory process of the pancreas with variable involvement of other regional tissues or remote organ systems

Diagnosis

The diagnosis of acute pancreatitis is secured when at least two of these three criteria are met

- The patients presenting signs and symptoms re consistent with the diagnosis
- The serum amylase or lipase is elevated
- CT scans demonstrate typical radiographic findings

Etiology

- Alcohol (the first episode of pancreatitis is usually preceded by 6 to 8 years of heavy alcohol ingestion ‹ recurring acute attacks ‹damaged pancreatic ductal system‹ chronic pancreatitis.‹high protein secretions‹ block small pancreatic ductules)
- Ingestion and biliary calculi account for 85% of cases of acute pancreatitis
- Metabolic, Mechanical, Postoperative, Traumatic, Vascular, Infectious, Genetic, and Autoimmune
- Obstruction in pancreatic ducts(gallstones (60% of nonalcoholic patients), tumors, trauma, and parasitic diseases)
- Choledocholithiasis (reflux of bile into the pancreatic duct or increased pancreatic ductal pressures)

Etiology

- Post-ERCP(1-5%)
- Idiopathic pancreatitis(8% to 10%)
- Biliary tract sludge (microlithiasis)
- Congenital causes (pancreatic divisum)
- Autoimmune IgG4 pancreatitis

The Atlanta Classification system

TABLE 17-1 Definition of the Grades of Severity of Acute Pancreatitis	
Term	**Definition**
Mild acute pancreatitis	No local complications No systemic complications
Moderately severe acute pancreatitis	Moderate local complications that resolve (acute fluid collections causing pain, fever, or an inability to eat) Transient organ failure of less than 48 hr
Severe acute pancreatitis	Intense local complications (necrosis, infected necrosis, pseudocysts) Persistent MSOF
Local Peripancreatic Complications of Acute Pancreatitis	
Acute fluid collections	Ill-defined collection of sterile fluid located in or about the pancreas, appearing early in the course of acute pancreatitis; lack of a wall of granulation of fibrous tissue; spontaneous regression usually occurs; if it persists, it develops into a pancreatic abscess or pseudocyst
Pancreatic necrosis	Diffuse or focal area(s) of nonviable pancreatic parenchyma typically associated with peripancreatic fat necrosis; nonenhanced pancreatic parenchyma
Acute pseudocyst	Collection of pancreatic juice enclosed by a wall of fibrous granulation tissue, which arises as a result of acute pancreatitis, pancreatic trauma, or chronic pancreatitis, occurring at least 4 wk after the onset of symptoms, is round or ovoid and most often sterile; when pus is present, the lesion is termed a *pancreatic abscess*.
Pancreatic abscess	Circumscribed, intra-abdominal collection of pus, usually in proximity to the pancreas, containing little or no pancreatic necrosis, which arises as a consequence of acute pancreatitis or pancreatic trauma; often 4 wk or more after onset; pancreatic abscess and infected pancreatic necrosis differ in clinical expression and in the extent of associated necrosis.

Clinical Presentation and Evaluation

- Noncrampy, constant, severe epigastric abdominal pain(radiates to the back)
- The pain may be alleviated by sitting or standing
- It is associated with nausea and vomiting
- Fever, tachycardia, and upper abdominal tenderness with guarding
- Dynamic ileus with abdominal distention
- Generalized abdominal and rebound tenderness may also occur in severe pancreatitis
- Retroperitoneal bleeding(Grey Turner’s sign - Cullen’s sign)
- SIRS (injury to other organs)

Laboratory Evaluation

- leukocytosis
- Elevated serum amylase(1.5) and lipase(5)
- Ultrasonography
- A chest x-ray
- Plain and upright abdominal x-rays
- In most cases, a CT scan is not required
- Pancreatic tissue that does not enhance with intravenous (IV) contrast is diagnostic of pancreatic necrosis (not an indication for surgical intervention)
- MRCP may benefit

TABLE 17-3 List of Disease Processes That May Result in Hyperamylasemia

Perforated Ulcer	Ovarian Tumor or Cyst
Ischemic bowel	Lung cancer
Small bowel obstruction	Prostate cancer
Renal failure	Diabetic ketoacidosis
Salivary gland infection	Macroamylasemia
Ectopic pregnancy	

The Differential Diagnosis

- Acute cholecystitis
- Perforated peptic ulcer
- Acute mesenteric ischemia
- Esophageal perforation
- Myocardial infarction

Prognosis

- A patient's outcome is directly related to the severity of the pancreatitis and the SIRS reaction
- Ranson grading system
- Five variables are measured on admission, and six additional variables are measured over the ensuing 48 hours
- The presence of three or more criteria indicates severe pancreatitis and is associated with an increased incidence of local and systemic complications
- Neither the serum amylase nor lipase are included in Ranson criteria, and hence neither is reflective of the severity of the pancreatitis nor the likelihood of developing complications; they are only markers of acinar cell damage

TABLE 17-4 Ranson's Criteria—Prognostic Factors for Major Complications or Death

	Nonbiliary	Biliary
On Admission		
Age	>55	>70
WBC count	>16	>18
Glucose	>200 mg/100 mL	>220 mg/100 mL
LDH	>350	>400
SGOT (AST)	>250	>250
During the Initial 48 hr		
Hematocrit decrease	>10%	>10%
BUN increase	>5 mg/dL	>2 mg/dL
Calcium	<8 mg/dL	<8 mg/dL
Arterial PO_2	<60 mm Hg	—
Base deficit	>4 mEq/L	>5 mEq/L
Fluid sequestration	>6 L	>4 L

Prognosis

- CT scans
- Acute pancreatitis is not a static disease, so a single CT scan at any one point in time might not truly reflect the severity of the disease at another point in time
- Serial CT scans are necessary to monitor the patient for surgically correctable complications

TABLE 17-5 CT Grading System for Acute Pancreatitis

Grade	CT Finding
A	Normal pancreas
B	Pancreatic enlargement
C	Pancreatic inflammation and/or peripancreatic fat
D	Single peripancreatic fluid collection
E	Two or more fluid collections and/or retroperitoneal air

Treatment

- General supportive therapy and the specific treatment
- Ranson score of >2: maintain adequate tissue perfusion and maintaining adequate intravascular volume
- Several liters of isotonic solution
- Use of a urinary catheter, central venous line, or cardiac echocardiogram
- Pulse oximetry

Treatment

- Intubation and aggressive ventilatory support
- All pharmacologic attempts have not demonstrated any significant benefit
- Nasogastric suction
- Prophylactic antibiotic use in severe disease
- Nutritional support
- enteral nutrition is preferred over parenteral nutrition

Surgical

- Prevent further episodes of pancreatitis
- Deal with local peripancreatic complications
- Cholecystectomy
- ERCP with sphincterotomy and stone extraction is advisable only in patients with severe pancreatitis and a suspected gallstone impacted at the ampulla of Vater(cholangitis or a persistently rising amylase)
- Early operative intervention in patients with sterile pancreatic necrosis carries prohibitive risks(not to operate at least 2-3weeks after its appearance)
- Surgical intervention(infected pancreatic necrosis or surgical complications in other organs)

Complications

Local:

- persistent or worsening abdominal pain and increases in serum amylase.
- Common bile duct obstruction
- Gastric outlet obstruction(treated by nasogastric decompression, fluid and electrolyte repletion, and possible surgical intervention)
- Splenic and/or portal vein (PV) thrombosis(treatment is splenectomy)

Complications

systemic complications:

- Function of the cytokine storm and the consequential SIRS response
- ARDS, acute kidney injury, and cardiovascular instability

TABLE 17-6 Systemic Complications Associated with Severe Pancreatitis	
Shock	Systolic blood pressure < 90 mm Hg
Pulmonary insufficiency	PaO_2/FiO_2 < 300
Renal failure	Creatinine ≥177 μmol/L or>2 mg/dL after rehydration
Gastrointestinal bleeding	500 mL in 24 hr
Disseminated intravascular coagulation	Platelets ≤100,000/mm^3, fibrinogen <1.0 g/L, and fibrin split products >80 μg/L
Severe metabolic disturbances	Calcium ≤ 1.87 mmol/L or ≤7.5 mg/dL

Infected Pancreatic Necrosis

- 20% of patients with acute pancreatitis
- The number one determinant of mortality(40%)
- The risk of infection is directly related to the extent of necrosis and usually occurs 2 to 3 weeks after the onset of severe necrotizing pancreatitis.
- A CT scan demonstrating edema surrounding the pancreas and demonstrating retroperitoneal air or air within the lesser sac

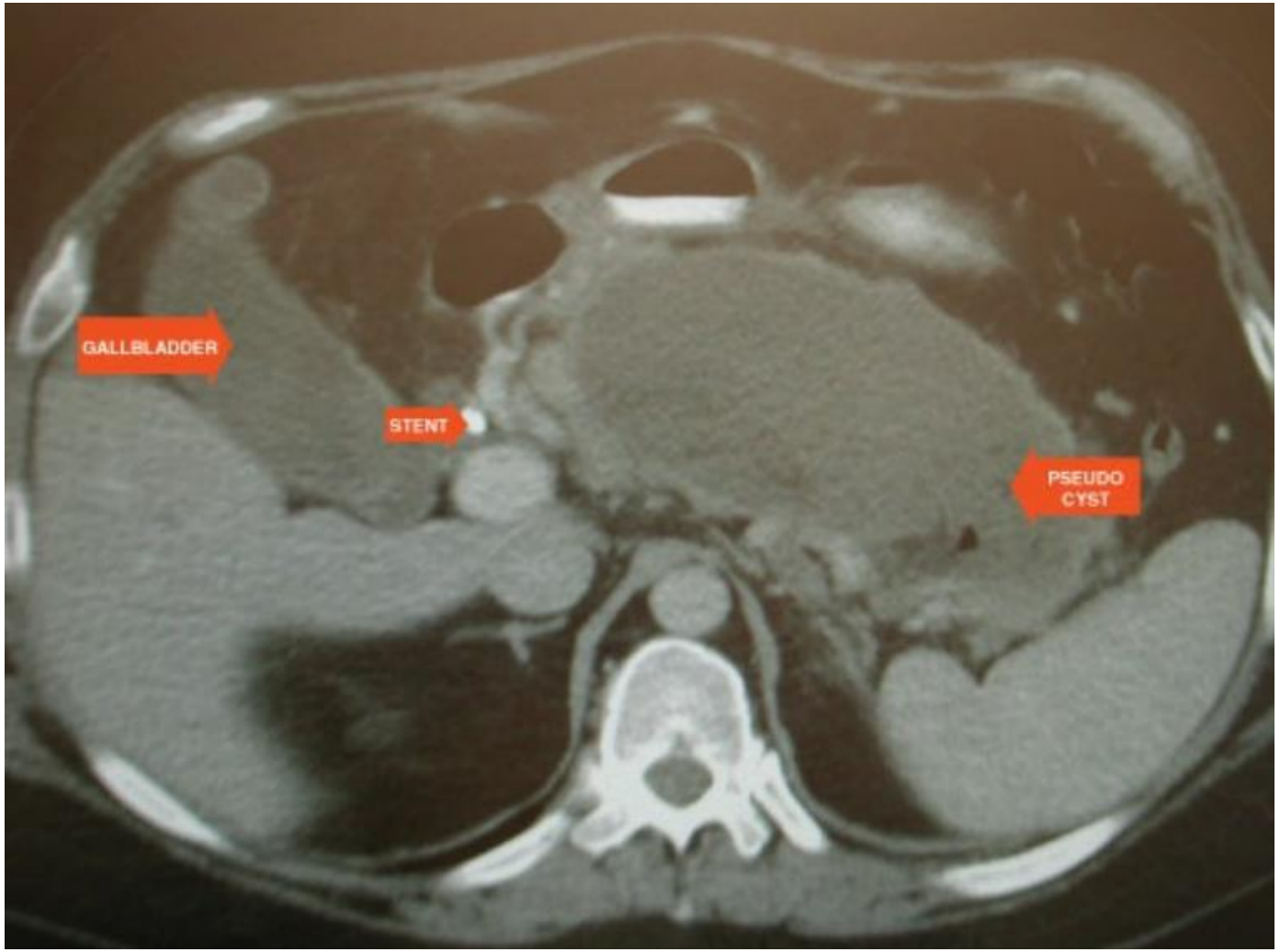
Edema and inflammation surrounding the majority of the pancreas

Peripancreatic Fluid Collections

- The most common complication
- This complication Is caused by disruption of the pancreatic duct and the leakage of activated pancreatic enzymes into the mesentery and retroperitoneum causing edema formation
- These patients are at risk of developing complications specific to the collection location and therefore should be followed closely for symptoms of biliary obstruction or gastric outlet obstruction(resolve spontaneously)

Pseudocysts

- Typically appearing some 3 to 4 weeks after onset of the acute pancreatitis
- Pseudocysts may be communicating or noncommunicating(mass effect)
- CT scans are the best imaging studies for pseudocyst evaluation
- External drainage of a communicating pseudocyst is contraindicated
- Noncommunicating pseudocysts may be aspirated or drained percutaneously with little risk of reformation or fistula formation


GALLBLADDER
STENT
PSEUDO
CYST

Chronic Pancreatitis

Etiology:

- Alcohol consumption
- anatomic variations
- cystic fibrosis

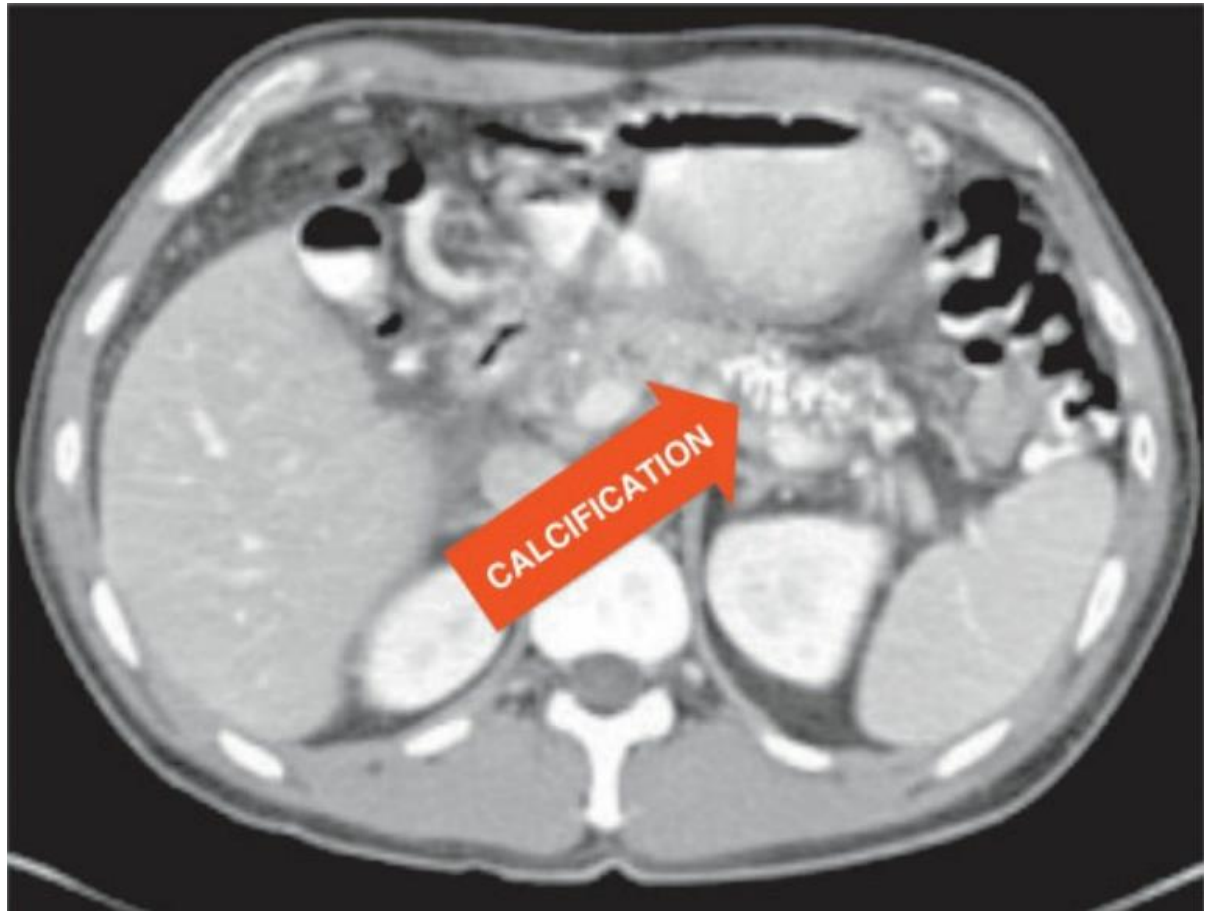
Clinical Presentation and Evaluation:

- Chronic pain (intermittent and constant)
- Food often makes pain worse
- When 90% of the gland has been damaged or replaced with scar, endocrine and exocrine insufficiencies occur
- Malabsorption, diabetes, and deficiencies in fat-soluble vitamins, causing malnutrition

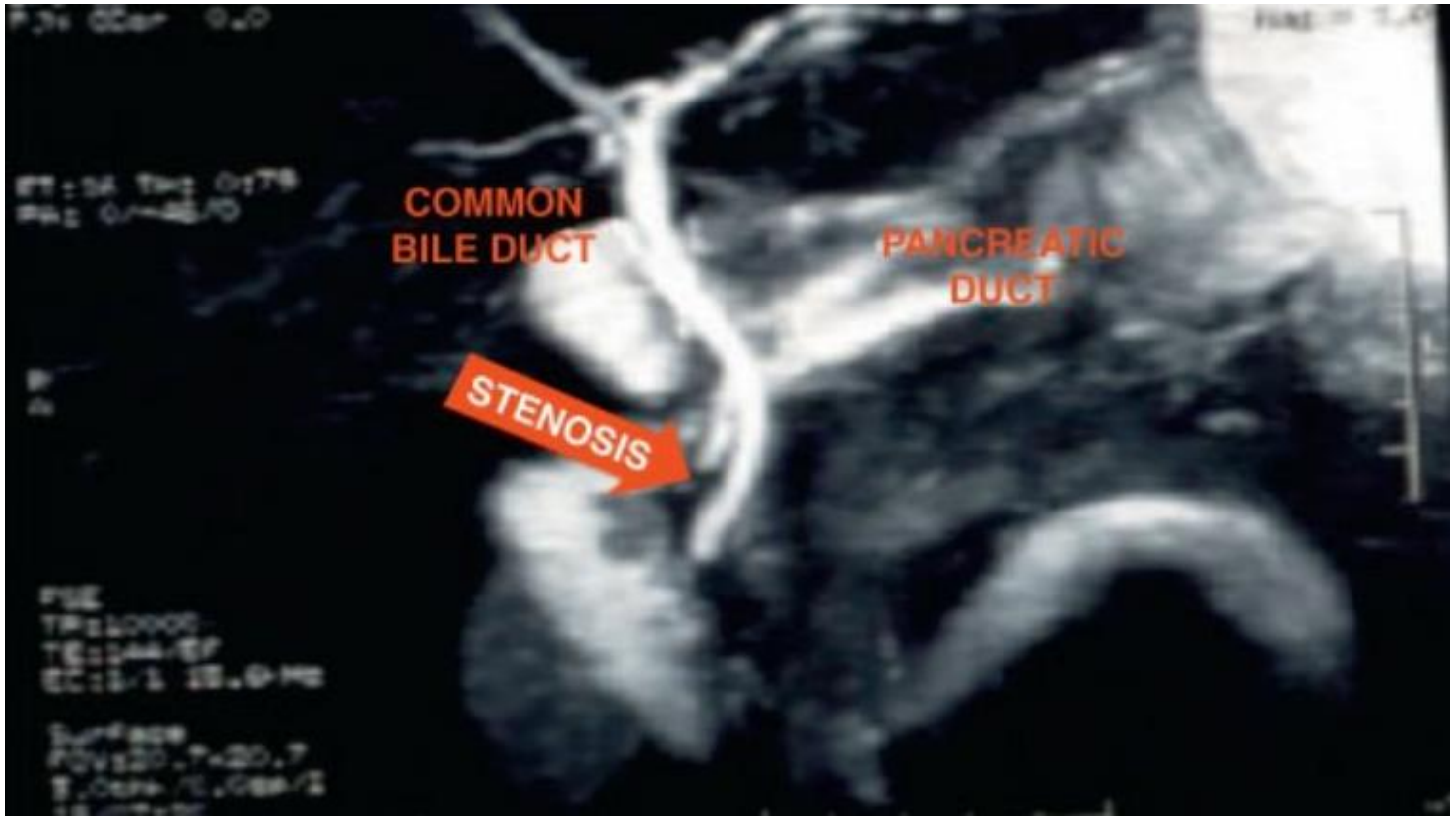
Chronic Pancreatitis

- The abdominal exam of these patients tends to be unremarkable
- Laboratory blood studies tend to be normal unless the chronic inflammation has led to biliary duct obstruction, in which case, typical lab values of obstructive jaundice are evident
- CT, MRCP, and ERCP may play a role in determining other non–alcohol-related causes

Multiple calcifications throughout the body and tail consistent with chronic pancreatitis



MRCP demonstrating stenosis of the minor duct and a dilated main pancreatic duct



Treatment

- Treatment of alcoholism
- Narcotic dependence and a low fat diabetic diet
- Pancreatic enzyme replacement to minimize steatorrhea and hyperglycemia
- Chronic pain: Surgery
- Surgical options for treating chronic pancreatitis fall into two main categories: drainage or resectional procedures

Surgical treatment

Drainage procedures:

- Patients with a dilated pancreatic duct(chain of lakes)
- Preserving any remaining functional pancreatic tissue, thereby delaying the onset of both exocrine and endocrine insufficiency
- lateral pancreaticojejunostomy (Puestow procedure)

Surgical treatment

Resectional procedures:

- Patients with nondilated ducts and disease that can be localized to a specific location
- Pancreaticoduodenectomy, distal pancreatectomy, and duodenal-preserving pancreatic head resections (Beger or Frey procedures)

Surgical treatment

- Total pancreatectomy has been utilized, but is not recommended(severe exocrine and endocrine dysfunction that follows
- Another option for pain relief in this group may be attempted via splanchnicectomy (neurolysis), which can be done percutaneously, endoscopically, or surgically, but results have been disappointing, with poor long-term durability

References

- JAMA 2004; 291:2865.
- UpToDate: Acute Pancreatitis – Clinical Features & Diagnosis.
- Br J Surg 2010; 97:1379

Thank you for your attention!