Pancreatitis, Acute

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INTRODUCTION

Background: According to the 1992 International Symposium on Acute Pancreatitis, acute pancreatitis is defined as an acute inflammatory process of the pancreas with variable involvement of other regional tissues or remote organ systems. Acute pancreatitis is classified further into mild and severe forms. Mild acute pancreatitis is associated with minimal organ dysfunction and uneventful recovery. Severe acute pancreatitis is associated with pancreatic necrosis and may lead to organ failure and/or local complications.

Local complications of acute pancreatitis include fluid collections, pseudocyst formation, abscess, pancreatic necrosis, hemorrhage, venous thrombosis, and pseudoaneurysm formation. A pseudocyst is defined as a collection of pancreatic juice enclosed by a wall of fibrous or granulation tissue. A pseudocyst lacks a true epithelial lining and often communicates with the pancreatic duct. A pancreatic abscess is a circumscribed intra-abdominal collection of pus. The development of both pseudocyst and abscess usually requires 4 or more weeks from the initial clinical onset of acute pancreatitis. Pancreatic necrosis is defined as focal or diffuse areas of nonviable pancreatic parenchyma, which usually is associated with peripancreatic fat necrosis. Necrosis usually develops early in the course of acute pancreatitis.
Gallstones and alcohol abuse are the most common causes of acute pancreatitis, accounting for 60-80% of cases. Other causes include blunt trauma to the abdomen, iatrogenic trauma (postoperative trauma, endoscopic retrograde cholangiopancreatography), hypertriglyceridemia, hypercalcemia, drug-induced, infectious etiologies (eg, mumps, cytomegalovirus), congenital anomalies (pancreas divisum, choledochocoele), ampullary or pancreatic tumors, vascular abnormalities (atherosclerotic emboli, hypoperfusion, vasculitis), cystic fibrosis, and Reye syndrome. These miscellaneous causes account for approximately 10% of cases of acute pancreatitis. In approximately 10-25% of patients, no underlying cause is found.

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Pathophysiology: The pathophysiology of acute pancreatitis is not understood clearly. Steinberg and Tenner note that a number of factors can initiate the inflammation, including but not limited to obstruction or overdistension of the pancreatic duct, exposure to ethanol and other toxins, hypertriglyceridemia, and hypercalcemia. The end result is a release of activated pancreatic destructive enzymes into the pancreas and surrounding tissues. As stated by Steinberg and Tenner, "an unpredictable cascade of events" ensues that results in mild localized interstitial pancreatitis or in severe necrosis. Furthermore, release of toxic factors (resulting from inflammatory response) into the systemic circulation may result in systemic complications such as shock, renal failure, or adult respiratory distress syndrome.

Frequency:

- **In the US:** Frequency of acute pancreatitis increases with age. According to Go, the rate is 270 cases per 100,000 in the United States in persons aged 15-44 years. The rate is 540 cases per 100,000 in persons older than 65 years.

Mortality/Morbidity: The clinical severity of acute pancreatitis varies from a mild uneventful course to a severe life-threatening illness associated with multisystemic organ failure (eg, shock, renal failure, adult respiratory distress syndrome). Multiple clinical systems have been devised to assess the severity and predict the prognosis of acute pancreatitis. These include the Ranson criteria, the modified Glasgow criteria, and acute physiology and chronic health evaluation (APACHE II) criteria.

Acute pancreatitis takes a mild course in approximately 70-80% of patients. According to Beger et al, most patients with acute pancreatitis suffer edematous interstitial pancreatitis, which is a mild self-limited disease. Approximately 20-30% of patients have a more severe clinical course. Pancreatic infection and multisystemic organ failure are the primary contributors to morbidity and mortality. More than 80% of deaths resulting from acute pancreatitis are from septic complications as a consequence of bacterial infection of pancreatic necrosis.

Race: For unknown reasons, the rate of pancreatitis in black Americans is 3 times higher than in white Americans. Maxson et al have reported that the prevalence of acute pancreatitis among patients with AIDS is approximately 4-22 per 100 patients. This is believed to result from increased infections of the pancreas and from HIV/AIDS medications.

Sex: Frequency of pancreatitis is approximately equal in men and women. Female patients tend to have pancreatitis caused by biliary stones, while alcohol abuse is the usual cause of pancreatitis in men.
Age: Pancreatitis is uncommon in children, with a rate of 2.7 cases per 100,000 in children younger than 15 years. The leading cause of acute pancreatitis in children is blunt trauma, which occasionally is associated with child abuse.

Anatomy: The pancreas is a retroperitoneal organ and is positioned in the anterior pararenal space. It is posterior to the stomach and lesser sac and anterior to the abdominal aorta and upper lumbar vertebrae. The pancreas is divided descriptively into 4 parts including the (1) head (which includes the uncinate process), (2) neck, (3) body, and (4) tail.

The head of the pancreas is nestled in the duodenal C-loop. The uncinate process curves around the superior mesenteric vein. The neck, body, and tail extend obliquely and superiorly where the tail is associated closely with the splenic hilum. The splenic vein is applied to the posterior border of the pancreas. The splenic vein merges with the superior mesenteric vein behind the pancreatic neck to form the portal vein confluence. The splenic artery and the gastroduodenal artery run along the superior and anterior surface of the pancreas, respectively. The common bile duct extends inferiorly through or behind the pancreatic head on its course to the duodenum. The pancreas also may have an ectopic location within the duodenal or gastric wall that can become inflamed as well.

The pancreatic exocrine secretions/enzymes primarily are drained by the duct of Wirsung, which extends the length of the gland. The duct of Wirsung may empty into the duodenal papilla separately or be joined by the common bile duct to form a common channel, which then empties into the duodenal papilla. An accessory duct of Santorini, located in the pancreatic head and neck, also is present and normally drains into the duodenum (just proximal to the duct of Wirsung).

Clinical Details: Typical symptoms include the sudden onset of epigastric abdominal pain that radiates to the back and flanks. The pain usually is constant and boring. Nausea and vomiting commonly are associated symptoms. The symptoms may begin after a heavy meal or after a drinking binge.

On physical examination, the abdomen may be distended and tenderness may exist over the upper abdomen. Rarely, the skin overlying the flank or abdominal wall may have a purple hue from hemorrhage (Grey Turner/Cullen sign). Common laboratory abnormalities include elevated serum amylase and lipase levels, hyperglycemia, hypocalcemia, decreased lactic dehydrogenase levels, elevated serum glutamic-oxaloacetic transaminase levels, and leukocytosis.

Preferred Examination: Contrast-enhanced computed tomography (CECT) is the imaging criterion standard for the evaluation of acute pancreatitis and its complications. Using non-contrast-enhanced CT, clinicians can establish the diagnosis and demonstrate fluid collections but cannot evaluate for pancreatic necrosis or vascular complications.

CECT allows complete visualization of the pancreas and retroperitoneum, even in the setting of ileus or overlying bandages from a recent surgical procedure. CECT can help detect almost all major abdominal complications of acute pancreatitis, such as fluid collections, pseudocysts, abscesses, venous thrombosis, and pseudoaneurysms. In addition, CECT can be used to guide percutaneous/interventional procedures such as diagnostic fine needle aspiration or catheter placement. CECT may be performed on severely ill patients including intubated patients. Lastly, CECT can be used as a prognostic indicator of the severity of acute pancreatitis.
Other adjunctive imaging modalities include ultrasound (US), MRI, and angiography. US is especially useful in the diagnosis of gallstones and follow-up observation of pseudocysts. US also can be used to detect pancreatic pseudoaneurysms. MRI has comparable diagnostic efficacy compared to CECT, although the examination is more time consuming and costly. Angiography primarily is used to help diagnose the vascular complications of acute pancreatitis.

**Limitations of Techniques:** The usefulness of CECT is limited in patients who have intravenous (IV) contrast allergy or renal insufficiency. Patients who have severe acute pancreatitis often require multiple scans to assess progress and/or complications. This necessitates significant radiation doses.

In addition, CECT is far less sensitive than US in detecting gallstones or biliary duct stones, a common cause of acute pancreatitis. Therefore, if gallstones or an impacted common bile duct stone are not seen on CT, US is necessary to document the presence or absence of gallstones.

**Differentials**

Other Problems to be Considered:

- Acute epigastric abdominal pain
- Peptic ulcer disease
- Gastritis
- Perforated viscus
- Esophageal spasm
- Intestinal obstruction
- Leaking aortic aneurysm

**Findings:** Plain films of the abdomen are part of the initial diagnostic workup of acute abdominal pain. The most commonly recognized radiologic signs associated with acute pancreatitis include the following:

- Air in the duodenal C-loop
- The sentinel loop sign, which represents a focal dilated proximal jejunal loop in the left upper quadrant
- The colon cutoff sign, which represents distention of the colon to the transverse colon with a paucity of gas distal to the splenic flexure

In a review of 73 cases by Rifkind et al, other plain film findings included obscuration of the psoas margin, increased epigastric soft tissue density, increased gastrocolic separation, gastric curvature distortion, pancreatic calcification, and pleural effusion (usually on left). Note that the abdominal plain film also can be completely normal in patients with acute pancreatitis.

**Degree of Confidence:** Findings on plain films are nonspecific but are suggestive of acute pancreatitis.
Findings: CECT of the abdomen and pelvis is the imaging criterion standard for evaluating acute pancreatitis and its complications. Both IV and oral contrast should be administered. Imaging protocols vary, but the most important unifying point is to obtain thin section images during the peak of pancreatic arterial perfusion. This usually can be acquired by imaging 30-40 seconds after the administration of iodinated contrast at 3-4 mL/s using helical CT. Some advocate the use of water as a negative contrast agent as barium in the duodenal sweep could potentially obscure a high attenuation stone.

Freeny recommends obtaining CECT in the following situations:

- Patients in whom the clinical diagnosis is in doubt
- Patients with hyperamylasemia and severe clinical pancreatitis, abdominal distention, tenderness, high fever, and leukocytosis
- Patients with a Ranson score greater than 3 or an APACHE score greater than 8
- Patients who do not manifest rapid clinical improvement within 72 hours of initiation of conservative medical therapy
- Patients who demonstrate clinical improvement during initial medical therapy but then manifest an acute change in clinical status, indicating a developing complication

Typical CT findings in acute pancreatitis include focal or diffuse enlargement of the pancreas, heterogeneous enhancement of the gland, irregular or shaggy contour of the pancreatic margins, blurring of peripancreatic fat planes with streaky soft tissue stranding densities, thickening of fascial planes, and the presence of intraperitoneal or retroperitoneal fluid collections. The fluid collections most commonly are found in the peripancreatic and anterior pararenal spaces but can extend from the mediastinum down to the pelvis.

Complications of acute pancreatitis, such as pseudocysts, abscess, necrosis, venous thrombosis, pseudoaneurysms, and hemorrhage, can be recognized with CECT.

- A pseudocyst appears as an oval or round water density collection with a thin or thick wall, which may enhance.
- A pancreatic abscess can manifest as a thick-walled low-attenuation fluid collection with gas bubbles or a poorly defined fluid collection with mixed densities/attenuation. Gas bubbles are not specific for infection and the diagnosis of a pancreatic abscess usually requires percutaneous fine needle aspiration to confirm the presence of pus.
- Necrotic pancreatic tissue is recognized by its failure to enhance after IV contrast administration. Balthazar et al point out that the normal unenhanced pancreas has CT attenuation numbers of 30-50 Hounsfield units (HU) and that after IV contrast, the pancreas should display attenuation numbers of 100-150 HU. A focal or diffuse well-marginated zone of unenhanced parenchyma (>3 cm in diameter or >30% of pancreatic area) is considered a reliable CT finding for the diagnosis of necrosis. Note that pancreatic necrosis may be radiologically indistinguishable from a pancreatic abscess.
- Venous thrombosis can be identified as a failure of the peripancreatic vein (eg, splenic vein, portal vein) to enhance or as an intraluminal filling defect.
- Associated gastric varices may be identified.
A pseudoaneurysm usually appears as a well-defined round structure with a contrast-enhancement pattern similar to the aorta and other arteries. Hemorrhage appears as high-attenuation fluid collections. Active bleeding is seen as contrast extravasation.

CECT can be used to assess the severity and estimate the prognosis of acute pancreatitis. Balthazar et al developed a grading system in which patients with acute pancreatitis are classified into 1 of the following 5 grades:

- Grade A - Normal-appearing pancreas
- Grade B - Focal or diffuse enlargement of the pancreas
- Grade C - Pancreatic gland abnormalities associated with peripancreatic fat infiltration
- Grade D - A single fluid collection
- Grade E - Two or more fluid collections

Patients with grades A and B pancreatitis have been shown to have mild uncomplicated clinical courses, while most complications of acute pancreatitis occur in patients with grades D and E.

Balthazar et al further constructed a CT severity index (CTSI) for acute pancreatitis that combines the grade of pancreatitis with the extent of pancreatic necrosis. The CTSI assigns points to patients according to their grade of acute pancreatitis as well as the degree of pancreatic necrosis. More points are given for a higher grade of pancreatitis and for more extensive necrosis. Patients with a CTSI of 0-3 had a mortality of 3% and a complication rate of 8%. Patients with a CTSI of 4-6 had a mortality rate of 6% and a complication rate of 35%. Patients with a CTSI of 7-10 had a 17% mortality rate and a 92% complication rate.

Grade of acute pancreatis and the points assigned per grade are as follows:

- Grade A - 0 points
- Grade B - 1 point
- Grade C - 2 points
- Grade D - 3 points
- Grade E - 4 points

Grade of necrosis and the points assigned per grade are as follows:

- None - 0 points
- Grade 0.33 - 2 points
- Grade 0.5 - 4 points
- Grade higher than 0.5 - 6 points

**Degree of Confidence:** In a prospective study of 202 patients, Clavien et al reported a 92% sensitivity and 100% specificity in diagnosing acute pancreatitis via CECT. Balthazar et al report an overall accuracy of 80-90% in the detection of pancreatic necrosis. Small areas of necrosis involving less than 30% of the pancreas can be missed. Nevertheless, the extent of pancreatic necrosis has been found to correlate well with operative findings and clinical severity. In a study by Block et al, the positive predictive value of CECT for pancreatic necrosis was found to be 92%.
False Positives/Negatives: The pancreas may appear normal in approximately 25% of patients with mild pancreatitis. In the acute phase of pancreatitis a small number of patients will have a false-positive diagnosis for necrosis due to massive interstitial edema and vasoconstriction of the vascular arcades. Repeat CT within a few days may show normal pancreatic enhancement.

Findings: Although CT has long been the mainstay for imaging acute pancreatitis and its complications, MRI is an excellent alternative imaging modality. Consider MRI as a viable alternative in situations in which CECT is contraindicated, such as in patients with contrast allergy or renal insufficiency.

- In addition to T1-weighted and fast spin-echo T2-weighted sequences, 2-dimensional Fourier transform (FT) and 3-dimensional FT gradient-echo sequences can be acquired rapidly to image the pancreas during patient breath holds, which reduces the artifacts related to physiologic motion.
- Bolus contrast administration of gadolinium chelates can be used to assess for pancreatic necrosis. The quality of upper abdominal imaging is enhanced further using phased-array surface coils and fat-suppression techniques.
- Detrimental effects of physiologic motion can be reduced further using ultrafast T2-weighted sequences, such as single-shot fast spin-echo or half-Fourier acquisition single-shot turbo-spin echo (HASTE) sequences. Subsecond image acquisitions provide diagnostic image quality even in the setting of uncooperative or tachypneic patients. These sequences also are used routinely for depicting the biliary tract in magnetic resonance cholangiopancreatography (MRCP).
- The normal pancreas demonstrates relatively high signal intensity on T1-weighted images with fat suppression.
- The morphologic changes of acute pancreatitis are similar on both CT and MRI.
- The pancreas may be enlarged focally (usually the pancreatic head) or diffusely.
- Acute inflammatory changes appear as low signal intensity strands in the surrounding peripancreatic fat.
- Complications of acute pancreatitis also can be identified. Hemorrhage is characterized by T1 shortening or high signal on T1-weighted sequences with fat suppression. Peripancreatic fluid collections, pseudocysts, and abscesses are recognized by their high signal intensity on T2-weighted sequences. Devascularized or necrotic portions of the pancreas fail to enhance on dynamic gadolinium-enhanced images. MRI also may be better than CT in detecting areas of sterile pancreatic necrosis in what appear to be simple pseudocysts on CT.

Degree of Confidence: In a small study by Saifuddin et al, MRI was found to be equivalent to CECT in helping assess the location and extent of peripancreatic inflammatory changes and fluid collections. In addition, MRI was found to be equivalent in helping assess the degree of pancreatic necrosis. Chalmers et al showed that MRI is more effective than CECT in helping characterize the content of fluid collections and in helping demonstrate gallstones.

False Positives/Negatives: Mild cases of acute pancreatitis can appear completely normal on MRI. MRI also is limited in detecting gas and calcifications.
Findings: Jeffrey recommends obtaining images of the pancreas and the peripancreatic compartments, such as the lesser sac, anterior pararenal space, and transverse mesocolon, by scanning in the supine, longitudinal, transverse, semi-erect, and coronal planes. However, regions of the pancreas may not be visible by US because of overlying bowel gas.

- The spleen can be used as an acoustic window to image the pancreatic tail.
- Laing et al further advise radiologists to scrutinize the intrapancreatic portion of the common bile duct carefully for biliary stones.
- Use Doppler techniques to assess vascular complications of acute pancreatitis such as venous thrombosis and pseudoaneurysm formation.
- US also is the most sensitive modality for concomitantly evaluating the biliary tree/gallbladder.
- The pancreas may appear sonographically normal in acute pancreatitis, especially in mild cases.
- More definitive findings include a diffusely enlarged hypoechoic gland.
- Focal enlargement of the pancreatic head and body also may be seen.

Complications of acute pancreatitis also may be identified.

- Peripancreatic free fluid collections are identified as ill-defined anechoic collections. The fluid collections may demonstrate internal echoes/debris or septations if hemorrhage or a superimposed infection has occurred.
- Extrapancreatic spread of acute pancreatitis may be the only sonographic manifestation of acute pancreatitis in some patients.
- Pseudocysts appear as well-defined round or oval anechoic fluid collections with through transmission. Infected and noninfected pseudocysts are indistinguishable from each other sonographically.
- US often is used to monitor the resolution of pancreatic pseudocysts.
- A pancreatic abscess may appear as a complex cystic structure with internal debris/septations and, possibly, echogenic gas bubbles.
- A pseudoaneurysm often appears as a cystic mass with turbulent arterial flow within the mass.
- Acute hemorrhage may be identified as a hyperechoic fluid collection.
- Venous thrombosis can be identified as an intraluminal filling defect.
- Associated gastric varices may be appreciated.

Degree of Confidence: A primary limitation of US is that often the pancreas cannot be visualized secondary to overlying bowel gas. Neoptolemos et al report a sensitivity of 67% and a specificity of 100% in the diagnosis of acute pancreatitis by US.

False Positives/Negatives: The pancreas may appear completely normal in mild cases of acute pancreatitis.
**Findings:** Vascular complications of acute pancreatitis result from the proteolytic effects of the pancreatic enzymes that cause erosion of blood vessels, which often results in pseudoaneurysm formation or free rupture. The splenic artery, followed by the pancreaticoduodenal and gastroduodenal arteries, are affected most commonly. The left gastric, hepatic, and small intrapancreatic arteries are involved less often.

If acute hemorrhage or pseudoaneurysm is suspected or diagnosed by US or CECT, a celiac/superior mesenteric arteriogram should be performed to definitively assess the extent of vascular involvement. In addition, permanent or temporary therapeutic embolization can be performed. The primary contraindication for angiography is a hemodynamically unstable patient.

The precise bleeding point is identified by noting free contrast extravasation. Once the site of pseudoaneurysm or source of active bleeding is identified, it can be treated by Gelfoam embolization, various coil occlusion devices, or tissue adhesives (eg, bucrylate). Superselective microcoil embolization also has been advocated by Reber et al. Vujic has suggested using small Gelfoam particles to control diffuse pancreatic surface bleeding. Diffuse bleeding from the gland may appear angiographically as a prominent blush.

Vujic purports that embolization also may be used as a temporizing measure to slow bleeding so that the patient may be operated on electively. This temporary therapeutic procedure involves selective or nonselective Gelfoam embolization or balloon occlusion of the main celiac trunk.

Complications of celiac/superior mesenteric arteriography and embolization include arterial injury such as thrombosis, dissection, or rupture, distal embolization, ischemia of visceral organs such as the spleen and bowel, coil malpositioning, and rebleeding.

Venous thrombosis of the splenic vein and/or collateral venous pathways also may be diagnosed via selective angiography.

**Degree of Confidence:** Precise identification of the pseudoaneurysm or bleeding site is crucial for effective treatment. Gambiez et al and Boudghene et al have reported sensitivities of 93% and 96%, respectively, in identifying the bleeding site. Success rates of 79% and 78% have been reported by Mandel et al and Boudghene et al, respectively, in embolizing pancreatic pseudoaneurysms.

**False Positives/Negatives:** Koehler et al have noted that failure to identify the bleeding source may be because of intermittent arterial bleeding, bleeding from a larger surface area, and venous bleeding. Gambiez et al have reported improper identification of the bleeding artery, inability to catheterize the bleeding vessel selectively, and inexperience of the angiographer as causes of embolization failure.

**Intervention:** CT and US are the guidance modalities of choice in performing diagnostic fine needle aspirations and percutaneous drainages of fluid collections. Diagnostic fine needle aspiration is performed to distinguish infected from noninfected pseudocysts and to delineate pancreatic abscess from infected necrosis. Send the aspirate at once for Gram stain and subsequent aerobic, anaerobic, and fungal cultures. Treatment regimens for these entities differ.
Noninfected pseudocysts can be left untreated since approximately 50% resolve spontaneously, while infected pseudocysts (abscesses) must be drained. Pitchumoni and Agarwal recommend that, regardless of size and duration, asymptomatic noninfected pseudocysts can be observed safely provided that they are monitored carefully and are not enlarging. Intervention is mandatory only in the presence of symptoms or complications such as obstruction or infection. A pancreatic abscess may be drained percutaneously, while infected necrosis usually requires surgical debridement.

VanSonnenberg et al suggest beginning needle aspirations with a 22-gauge needle and then advancing to larger gauge needles if fluid cannot be withdrawn. Thicker fluid also requires larger and more numerous drains. Several techniques can be used for percutaneous catheter drainage. Catheter aftercare or monitoring is important. VanSonnenberg et al recommend saline irrigation of percutaneous catheters every 4-12 hours. Catheters usually are not removed until drainage ceases. This usually takes longer than in other abscesses. Sinograms through the catheter, abscessograms, or repeat CT or US examinations can be performed to assess resolution of the pseudocyst or abscess.

Medical/Legal Pitfalls:

- The primary complication of fine needle aspiration results from seeding bacteria into a sterile fluid collection. This is an unusual complication.

- Pitchumoni et al report that the primary complications of percutaneous catheter drainage include introduction of secondary infection or catheter-related infection, occlusion or displacement of the catheter, cellulitis at the site of entry, and accidental injury to other abdominal organs, such as the spleen.

- Less common complications include bowel perforation, GI hemorrhage, and fistula formation in the stomach, jejunum, or cecum.

Caption: Picture 1. Acute pancreatitis. Focal pancreatitis involving pancreatic head. Pancreatic head is enlarged with adjacent ill-defined peripancreatic inflammation and fluid collections

Caption: Picture 2. Acute pancreatitis. Pancreatic abscess. Large, relatively well-circumscribed heterogeneous collection containing gas bubbles inferior to the pancreatic head. This collection was drained successfully and percutaneously via a 12Fr pigtail catheter
Picture Type: MRI

Caption: Picture 3. Acute pancreatitis. Pancreatic necrosis. Note the nonenhancing pancreatic body anterior to the splenic vein. Also present is peripancreatic fluid extending anteriorly from the pancreatic head.

Picture Type: CT

Caption: Picture 4. Acute pancreatitis. Pancreatic necrosis. Approximately 50% of the pancreatic gland does not display enhancement after contrast administration.

Picture Type: MRI

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BIBLIOGRAPHY


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