ACUTE PANCREATITIS

(extraido de Papadakis et al. Current Medical Diagnosis and Treatment, Ed. 2017)

- Abrupt onset of deep epigastric p a i n , often with radiation to the back .
- H i story of previous episodes, often related to alcohol intake.
- Nausea, vomiting, sweating, weakness.
- Abdom i n a l tenderness and distention and fever.
- Leukocytosis, elevated serum amylase, elevated serum lipase.

General Considerations

The annual incidence of acute pancreatitis ranges from 13 to 45 per 1 00,000 population and has increased since 1 990. Most cases of acute pancreatitis are related to biliary tract disease (a passed gallstone, usually 5 mm or less in diameter) or heavy alcohol intake. The exact pathogenesis is not known but may include edema or obstruction of the ampulla of Vater, reflux of bile into pancreatic ducts, and direct injury of pancreatic acinar cells by prematurely activated pancreatic enzymes. Among the numerous other causes or associations are hypercalcemia, hyperlipidemias (chylomicronemia, hypertriglyceridemia, or both), abdominal

trauma (including surgery) , drugs (including azathioprine, mercaptopurine, asparaginase, p entamidine, didanosine, valproic acid, tetracyclines, dapsone, isoniazid, metronidazole, estrogen and tamoxifen [by raising serum triglycerides] , sulfonamides, mesalamine, celecoxib, sulindac, leflunomide, thiazides, simvastatin, fenofibrate, enalapril, methyldopa, procainamide, sitagliptin, exenatide,possibly corticosteroids, and others) , vasculitis, infections (eg, mumps, cytomegalovirus, M avium intracellulare complex), peritoneal dialysis, cardiopulmonary bypass, single or double-balloon enteroscopy, and ERCP. In patients with pancreas divisum, a congenital anomaly in which the dorsal and ventral pancreatic ducts fail to fuse, acute pancreatitis may result from stenosis of the minor papilla with obstruction to flow from the accessory pancreatic duct,although concomitant genetic mutations, particularly in

the cystic fibrosis transmembrane conductance regulator (CFTR) gene, have also been reported to account for acute pancreatitis in some patients with pancreas divisum. Genetic mutations also predispose to chronic pancreatitis, particularly in persons younger than 30 years of age if no other cause is evident and a family history of pancreatic disease is present. Acute pancreatitis may also result from the anomalous union of the pancreaticobiliary duct. Rarely, acute pancreatitis may be the presenting manifestation of a pancreatic or ampullary neoplasm. Celiac disease appears to be associated with an increased risk of acute and chronic

pancreatitis. Apparently "idiopathic" acute pancreatitis is often caused by occult biliary microlithiasis and may be caused by sphincter of Oddi dysfunction involving the pancreatic duct. Between 1 5 % and 25% of cases are truly idiopathic. Smoking, high dietary glycemic load, and abdominal adiposity increase the risk of pancreatitis, and older age and obesity increase the risk of a severe course; vegetable consumption and use of statins may reduce the risk of pancreatitis.

Clinical Findings

A. Symptoms and Signs

Epigastric abdominal pain, generally abrupt in onset, is steady, boring, and severe and often made worse by walking and lying supine and better by sitting and leaning forward. The pain usually radiates into the back but may radiate to the right or left. Nausea and vomiting are usually present. Weakness, sweating, and anxiety are noted in severe attacks. There may be a history of alcohol intake or a heavy meal immediately preceding the attack or a history of milder similar episodes or biliary pain in the past.

The abdomen is tender mainly in the upper part, most often without guarding, rigidity, or rebound. The abdomen may be distended, and bowel sounds may be absent with associated ileus. Fever of 38.4-39°C, tachycardia, hypotension (even shock) , pallor, and cool clammy skin are present in severe cases. Mild jaundice may be seen. Occasionally, an upper abdominal mass due to the inflamed pancreas or a pseudocyst may be palpated.

Acute kidney injury (usually prerenal) may occur early in the course of acute pancreatitis.

B. Laboratory Findings

Serum amylase and lipase are elevated-usually more than three times the upper limit of normal-within 24 hours in 90% of cases; their return to normal is variable depending on the severity of disease. Lipase remains elevated longer than amylase and is slightly more accurate for the diagnosis of acute pancreatitis. Leukocytosis (10,000-30,000/mcL), proteinuria, granular casts, glycosuria (1 0-20% of cases), hyperglycemia, and elevated serum bilirubin may be present.

Blood urea nitrogen and serum alkaline phosphatase may be elevated and coagulation tests abnormal. An elevated serum creatinine level (greater than 1 . 8 mg/Dl [149.94 mcmol/L]) at 48 hours is associated with the development of pancreatic necrosis. In patients with clear evidence of acute pancreatitis, a serum ALT level of more than 1 50 units/L (3 mkat/L) suggests biliary pancreatitis. A decrease in serum calcium may reflect saponification and correlates with severity of the disease. Levels lower than 7 mg/dL (1 .75 mmoi!L) (when serum albumin is normal) are associated with tetany and an unfavorable prognosis.

Patients with acute pancreatitis caused by hypertriglyceridemia generally have fasting triglyceride levels above 1 000 mg/dL (1 0 mmoi!L) ; in some cases, the serum amylase isnot elevated substantially because of an inhibitor in the serum of patients with marked hypertriglyceridemia that interferes with measurement of serum amylase. An early rise in the hematocrit value above 44% suggests hemoconcentration and predicts pancreatic necrosis. An elevated C-reactive protein concentration (greater than 150 mg/L [1 500 mg/L]) at 48 hours suggests severe disease.

Other diagnostic tests that offer the possibility of simplicity, rapidity, ease of use, and low cost-including urinary trypsinogen-2, trypsinogen activation peptide, and carboxypeptidase B-are not widely available. In patients in whom ascites or a left pleural effusion develops, fluid amylase content is high. Electrocardiography may show ST-T wave changes.

C. Assessment of Severity

In addition to the individual laboratory parameters noted above, the severity of acute alcoholic pancreatitis can be assessed using several scoring systems, including the Ranson criteria (Table 1 6-9). The Sequential Organ Failure Assessment (SOFA) score or modified Marshall scoring system can be used to assess injury to other organs, and the Acute Physiology and Chronic Health Evaluation (APACHE II) score is another tool for assessing severity. A simple 5-point clinical scoring system (the Bedside Index for Severity in Acute Pancreatitis, or BISAP) based on blood urea nitrogen above 25 mg/dL (9 mmol/L), impaired mental status, systemic inflammatory response syndrome, age older than 60 years, and pleural effusion during the first 24 hours (before the onset of organ failure) identifies patients at increased risk for mortality. More simply, the presence of a systemic inflammatory response alone and an elevated blood urea nitrogen level on admission as well as a rise in blood urea nitrogen within the first 24 hours of hospitalization are independently associated with increased mortality; the greater the rise in blood urea nitrogen after admission, the greater the mortality rate. A model based on the change in serum amylase in the first 2 days after admission and the body mass index has been proposed. An early rise in serum levels of neutrophil gelatinase-associated lipocalin has also been proposed as a marker of severe acute pancreatitis. The absence of rebound abdominal tenderness or guarding, a normal hematocrit value, and a normal serum creatinine level (the "harmless acute pancreatitis score;' or HAPS) predicts a nonsevere course with 98% accuracy. The revised Atlanta classification of the severity of acute pancreatitis uses the following three categories: (1) mild disease is the absence of organ failure and local ([peri] pancreatic necrosis or fluid collections) or systemic complications; (2) moderate disease is the presence of transient (under 48 hours) organ failure or local or systemic complications, or both; and (3) severe disease is the presence of persistent (48 hours or more) organ failure. A similar "determinant-based" classification includes a category of critical acute pancreatitis characterized by both persistent organ failure and infected peripancreatic necrosis.

D. Imaging

Plain radiographs of the abdomen may show gallstones (if calcified) , a "sentinel loop" (a segment of air-filled small intestine most commonly in the left upper quadrant), the "colon cutoff sign"-a gas-filled segment of transverse colon abruptly ending at the area of pancreatic inflammation-or focal linear atelectasis of the lower lobe of the lungs with or without pleural effusion. Ultrasonography is often not helpful in diagnosing acute pancreatitis because of intervening bowel gas but may identify gallstones in the gallbladder. Unenhanced CT is useful for demonstrating an enlarged pancreas when the diagnosis of pancreatitis is uncertain, differentiating pancreatitis from other possible intra-abdominal catastrophes, and providing an initial assessment of prognosis but is often unnecessary early in the course (Table 1 6 - 1 0) . Rapid-bolus intravenous contrast-enhanced CT following aggressive volume resuscitation is of particular value after the first 3 days of severe acute pancreatitis for identifying areas of necrotizing pancreatitis and assessing the degree of necrosis, although the use of intravenous contrast may increase the risk of complications of pancreatitis and of acute kidney injury and should be avoided when the serum creatinine level is above 1.5 mg/dL (1 24.95 mcmol/L). MRI appears to be a suitable alternative to CT. Perfusion CT on day 3 demonstrating areas of ischemia in the pancreas has been reported to predict the development of pancreatic necrosis.

The presence of a fluid collection in the pancreas correlates with an increased mortality rate. CT-guided needle aspiration of areas of necrotizing pancreatitis after the third day may disclose infection, usually by enteric organisms, which typically requires debridement. The presence of gas bubbles on CT implies infection by gas-forming organisms. EUS is useful in identifying occult biliary disease (eg, small stones, sludge, microlithiasis), which is present in a majority of patients with apparently idiopathic acute pancreatitis, and is indicated in persons over age 40 to exclude malignancy. ERCP is generally not indicated after a first attack of acute pancreatitis unless there is associated cholangitis or jaundice or a bile duct stone is known to be present, but EUS or MRCP should be considered, especially after repeated attacks of idiopathic acute pancreatitis. In selected cases, aspiration of bile for crystal analysis may confirm the suspicion of microlithiasis, and manometry of the pancreatic duct sphincter may detect sphincter of Oddi dysfunction as a cause of recurrent pancreatitis.

Differential Diagnosis

Acute pancreatitis must be differentiated from an acutely perforated duodenal ulcer, acute cholecystitis, acute intestinal obstruction, leaking aortic aneurysm, renal colic, andacute mesenteric ischemia. Serum amylase may also be elevated in high intestinal obstruction, in gastroenteritis, in mumps not involving the pancreas (salivary amylase), in ectopic pregnancy, after administration of opioids, and after abdominal surgery. Serum lipase may also be elevated in many of these conditions.

Complications

Intravascular volume depletion secondary to leakage of fluids in the pancreatic bed and ileus with fluid-filled loops of bowel may result in prerenal azotemia and even acute tubular necrosis without overt shock. This sequence usually occurs within 24 hours of the onset of acute pancreatitis and lasts 8-9 days. Some patients require renal replacement therapy.

According to the revised Atlanta classification, fluid collections and necrosis may be acute (within the first 4 weeks) or chronic (after 4 weeks) and sterile or infected. Chronic collections, including pseudocysts and walled-off necrosis, are characterized by encapsulation. Sterile or infected necrotizing pancreatitis may complicate the course of 5 - 1 0 % of cases and accounts for most of the deaths. The risk of infection does not correlate with the extent of necrosis.

Pancreatic necrosis is often associated with fever, leukocytosis, and, in some cases, shock and is associated with organ failure (eg, gastrointestinal bleeding, respiratory failure, acute kidney injury) in 50% of cases. Because infected pancreatic necrosis is often an indication for debridement, fine-needle aspiration of necrotic tissue under CT guidance should be performed (if necessary, repeatedly) for Gram stain and culture. A serious complication of acute pancreatitis is acute respiratory distress syndrome (ARDS); cardiac dysfunction may be superimposed. It usually occurs 3-7 days after the onset of pancreatitis in patients who have required large volumes of fluid and colloid to maintain blood pressure and urinary output. Most patients with ARDS require intubation, mechanical ventilation, and supplemental oxygen.

Pancreatic abscess (also referred to as infected or suppurative pseudocyst) is a suppurative process characterized by rising fever, leukocytosis, and localized tenderness and an epigastric

mass usually 6 or more weeks into the course of acute pancreatitis. The abscess may be associated with a left-sided pleural effusion or an enlarging spleen secondary to splenic vein thrombosis. In contrast to infected necrosis, the mortality rate is low following drainage.

Pseudocysts, encapsulated fluid collections with high amylase content, commonly appear in pancreatitis when CT is used to monitor the evolution of an acute attack. Pseudocysts that are smaller than 6 em in diameter often resolve spontaneously. They most commonly are within or adjacent to the pancreas but can present almost anywhere (eg, mediastinal, retrorectal) by extension along anatomic planes. Multiple pseudocysts are seen in 1 4% of cases. Pseudocysts may become secondarily infected, necessitating drainage as for an abscess. Pancreatic ascites may present after recovery from acute pancreatitis as a gradual increase in abdominal girth and persistent elevation of the serum amylase level in the absence of frank abdominal pain. Marked elevations in ascitic protein (greater than 3 g/dL) and amylase (greater than 1 000 units/L [20 mkat/L]) concentrations are typical. The condition results from disruption of the pancreatic duct or drainage of a pseudocyst into the peritoneal cavity.

Rare complications of acute pancreatitis include haemorrhage caused by erosion of a blood vessel to form a pseudoaneurysm and colonic necrosis. Portosplenomesenteric venous thrombosis frequently develops in patients with necrotizing acute pancreatitis but rarely leads to complications.

Chronic pancreatitis develops in about 10% of cases. Permanent diabetes mellitus and exocrine pancreatic insufficiency occur uncommonly after a single acute episode.

Treatment

A. Treatment of Acute Disease

1 . Mild disease-In most patients, acute pancreatitis is a mild disease ("nonsevere acute pancreatitis") that subsides spontaneously within several days. The pancreas is "rested" by a regimen of withholding food and liquids by mouth, bed rest, and, in patients with moderately severe pain or ileus and abdominal distention or vomiting, nasogastric suction. Early fluid resuscitation (one- third of the total 72-hour fluid volume administered within 24 hours of presentation, 250-500 mL/h initially) may reduce the frequency of systemic inflammatory response syndrome and organ failure in this group of patients, and lactated Ringersolution may be preferable to normal saline; however, overly aggressive fluid resuscitation may lead to morbidity as well. Pain is controlled with meperidine, up to 100- 150 mg intramuscularly every 3-4 hours as necessary. In those with severe liver or kidney dysfunction, the dose may need to be reduced. Morphine had been thought to cause sphincter of Oddi spasm but is now onsidered an acceptable alternative and, given the potential side effects of meperidine, may even be preferable. Oral intake of fluid and foods can be resumed when the patient is largely free of pain and has bowel sounds (even if the serum amylase is still elevated) . Clear liquids are given first (this step may be skipped in patients with mild acute pancreatitis) , followed by gradual advancement to a low-fat diet, guided by the patient's tolerance and by the absence of pain. Pain may recur on refeeding in 20% of patients. Following recovery from acute biliary pancreatitis, laparoscopic cholecystectomy is generally performed, preferably during the same

hospital admission, and is associated with a reduced rate of recurrent gallstone-related complications compared with delayed cholecystectomy. In selected cases endoscopic sphincterotomy alone may be done. In patients with recurrent pancreatitis associated with pancreas divisum, insertion of a stent in the minor papilla (or minor papilla sphincterotomy) may reduce the frequency of subsequent attacks, although complications of such therapy are frequent. In patients with recurrent acute pancreatitis attributed to pancreatic sphincter of Oddi dysfunction, biliary sphincterotomy alone is as effective as combined biliary and pancreatic sphincterotomy in reducing the frequency of recurrent acute pancreatitis, but chronic pancreatitis may still develop in treated patients. Hypertriglyceridemia with acute pancreatitis has been treated with insulin, heparin, or apheresis, but the benefit of these approaches has not been proven.

2. Severe disease-In more severe pancreatitis-particularly necrotizing pancreatitis-there may be considerable leakage of fluids, necessitating large amounts of intravenous fluids (eg, 500 - 1 000 mL/h for several hours, then 250-300 mL!h) to maintain intravascular volume. Risk factors for high levels of fluid sequestration include younger age, alcohol etiology, higher hematocrit value, higher serum glucose, and systemic inflammatory response syndrome in the first 48 hours of hospital admission. Hemodynamic monitoring in an intensive care unit is required, and the importance of aggressive intravenous hydration targeted to result in adequate urinary output, stabilization of blood pressure and heart rate, restoration of central venous pressure, and a modest decrease in hematocrit value cannot be overemphasized.

Calcium gluconate must be given intravenously if there is evidence of hypocalcemia with tetany. Infusions of fresh frozen plasma or serum albumin may be necessary in patients with coagulopathy or hypoalbuminemia. With colloid solutions, the risk of ARDS may be increased. If shock persists after adequate volume replacement (including packed red cells) , pressors may be required. For the patient requiring a large volume of parenteral fluids, central venous pressure and blood gases should be monitored at regular intervals. Enteral nutrition via a nasojejunal or possibly nasogastric feeding tube is preferable to parenteral nutrition in patients who will otherwise be without oral nutrition for at least 7- 1 0 days and reduces the risk of multiorgan failure and mortality when started within 48 hours of admission, but may not be tolerated in some patients with an ileus and does not reduce the rates of infection and death compared with the introduction of an oral diet after 72 hours. Parenteral nutrition (including lipids) should be considered in patients who have severe pancreatitis and ileus; glutamine supplementation appears to reduce the risk of infectious complications and mortality.

The routine use of antibiotics to prevent conversion of sterile pancreatic necrosis to infected necrosis is still controversial and generally is not indicated in those with less than 30% pancreatic necrosis. Imipenem (500 mg every 8 hours intravenously) and possibly cefuroxime (1 . 5 g intravenously three times daily, then 250 mg orally twice daily) administered for no more than 14 days to patients with sterile pancreatic necrosis has been reported in some studies to reduce the risk of pancreatic infection and mortality; meropenem and the combination of ciprofloxacin and metronidazole do not appear to reduce the frequency ofinfected necrosis, multiorgan failure, or mortality. When infected necrosis is confirmed, imipenem or meropenem should be continued. In occasional cases, a fungal infection

is found, and appropriate antifungal therapy should be prescribed. The role of intravenous somatostatin in severe acute pancreatitis is uncertain, and octreotide is thought to have no benefit. A small study has suggested benefit from pentoxifylline. To date, probiotic agents have not been shown to reduce infectious complications of severe pancreatitis and may increase mortality. Nonsteroidal anti-inflammatory drugs (eg, indomethacin administered rectally) and aggressive hydration with lactated Ringer solution have been reported to reduce the frequency and severity of post- ERCP pancreatitis in persons at high risk, and rectal indomethacin has become standard practice.

There is some evidence that the risk of pancreatitis after ERCP can be reduced by the administration of somatostatin, octreotide, nafamostat and other protease inhibitors, ulinastatin, and nitroglycerin, but further studies are needed. Placement of a stent across the pancreatic duct or orifice has been shown to reduce the risk of post-ERCP pancreatitis and is a common practice but has not been compared directly with rectal indomethacin.

B. Treatment of Complications and Follow-Up

A surgeon should be consulted in all cases of severe acute pancreatitis. If the diagnosis is in doubt and investigation indicates a strong possibility of a serious surgically correctable lesion (eg, perforated peptic ulcer), exploratory laparotomy is indicated. When acute pancreatitis is found unexpectedly, it is usually wise to close without intervention. If the pancreatitis appears mild and cholelithiasis or microlithiasis is present, cholecystectomy or cholecystostomy may be justified. When severe pancreatitis results from choledocholithiasis and j aundice (serum total bilirubin above 5 mg/dL [85.5 mcmol/L]) or cholangitis is present, ERCP with endoscopic sphincterotomy and stone extraction is indicated. MRCP may be useful in selecting patients for therapeutic ERCP. Endoscopic sphincterotomy does not appear to improve the outcome of severe pancreatitis in the absence of cholangitis or jaundice. Necrosectomy may improve survival in patients with necrotizing pancreatitis and clinical deterioration with multiorgan failure or lack of resolution by 4 weeks and is often indicated for infected necrosis, although a select group of relatively stable patients with infected pancreatic necrosis may be managed with antibiotics alone. The goal is to debride necrotic pancreas and surrounding tissue and establish adequate drainage. Outcomes are best if necrosectomy is delayed until the necrosis has organized, usually about 4 weeks after disease onset. A "step-up" approach in which nonsurgical drainage of walled -off pancreatic necrosis under radiologic guidance with subsequent open surgical necrosectomy if necessary has been shown to reduce mortality and resource utilization in selected patients with necrotizing pancreatitis and confirmed or suspected secondary infection. Endoscopic (transgastric or transduodenal) drainage combined with percutaneous drainage and, in some cases, laparoscopic guidance are additional options, depending on local expertise. Treatment is labor intensive, and multiple procedures are often required. Peritoneal lavage has not been shown to improve survival in severe acute pancreatitis, in part because the risk of late septic complications is not reduced. The development of a pancreatic abscess is an indication for prompt percutaneous or surgical drainage. Chronic pseudocysts require endoscopic, percutaneous catheter, or surgical drainage when infected or associated with persisting pain, pancreatitis, or bile duct obstruction. For pancreatic infections, imipenem, 500 mg every 8 hours intravenously, is a good choice of

antibiotic because it achieves bactericidal levels in pancreatic tissue for most causative organisms. Pancreatic duct leaks and fistulas may require endoscopic or surgical therapy.

Prognosis

Mortality rates for acute pancreatitis have declined from at least 10% to around 5% since the 1 980s, but the mortality rate for severe acute pancreatitis (more than three Ranson criteria; Table 16-9) remains at least 20%, with rates of 10% and 25% in those with sterile and infected necrosis, respectively. Severe acute pancreatitis is predicted by features of the systemic inflammatory response on admission; a persistent systemic inflammatory response is associated with a mortality rate of 25% and a transient response with a mortality rate of 8%. Half of the deaths occur within the first 2 weeks, usually from multiorgan failure. Multiorgan failure is associated with a mortality rate of at least 30%, and if it persists beyond the first 48 hours, the mortality rate is over 50%. Later deaths occur because of complications of infected necrosis. The risk of death doubles when both organ failure and infected necrosis are present. Moreover, hospital-acquired infections increase the mortality of acute pancreatitis, independent of severity. Readmission to the hospital for acute pancreatitis within 30 days may be predicted by a scoring system based on five factors during the index admission: eating less than a solid diet at discharge; nausea, vomiting, or diarrhea at discharge; pancreatic necrosis; use of antibiotics at discharge; and pain at discharge. Male sex, an alcohol etiology, and severe acute disease are risk factors. Recurrences are common in alcoholic pancreatitis but can be reduced by repeated, regularly scheduled interventions to eliminate alcohol consumption. after discharge from the hospital. A severe initial attack and smoking also increase the risk of recurrence. The risk of chronic pancreatitis following an episode of acute alcoholic pancreatitis is 1 3 % in 10 years and 1 6% in 20 years, and the risk of diabetes mellitus is increased more than twofold over 5 years. Overall, chronic pancreatitis develops in 36% of patients with recurrent acute pancreatitis; alcohol use and smoking are principal risk factors.

When to Admit

Nearly all patients with acute pancreatitis should be hospitalized.

Table 16-9. Ranson criteria for assessing the severity of acute pancreatitis.

Three or more of the following predict a severe course complicated by pancreatic necrosis with a sensitivity of 60-80%

- Age over 55 years
- White blood cell count > 1 6 x 1 03/mcl (> 1 6 x 1 09/L)
- Blood glucose > 200 mg/d l (> 1 1 mmoi/L)
- Serum lactic dehydrogenase > 350 u n its/L (> 7 m kat/L)
- Aspartate aminotransferase > 250 u n its/L (> 5 mkat/L)

Development of the following in the first 48 hours indicates a worsening prognosis

- Hematocrit drop of more than 10 percentage points
- Blood urea nitrogen rise > 5 mg/dl (> 1 .8 m m o i/L)
- Arterial PO, of < 60 mm H g (< 7.8 kPa)
- Serum calcium of < 8 mg/dl (< 0.2 mmoi/L)
- Base deficit over 4 m Eq/L
- Estimated fluid sequestration of > 6 L

Mortality rates correlate with the number of criteria present'

Number of Criteria	Mortality Rate (%)
0-2	1,0
3-4	16,0
5-6	40,0
7-8	100,0

1 An APACHE II score > 8 also correlates with mortality.

Table 16-10. Severity index for acute pancreatitis.

CT Grade	*Points*	*Pancreatic Necrosis (%)*	*Additional Points*	*Severity Index'*	*Mortality Rate (2) (%)*
A Normal pancreas	0	0	0	0	0
B Pancreatic enlargement	1	0	0	1	0
C Pancreatic inflammation and/or peri pancreatic fat	2	<30	2	4	<3
D Single acute peri pancreatic fluid collection	3	30-50	4	7	6
E Two or more acute peri pancreatic fluid collections or retroperitoneal air	4	>50	6	10	>17

(1) - Severity Index = CT Grade Points + Pancreatic Necrosis Additional Points.

(2) - Based on the Severity Index.
Adapted with permission from Balthazar EJ Acute pancreatitis: assessment of severity with clinical and CT evaluation. Radiology. 2002; Jun;223 (3):603-13.