Metabolic problems, which are common in severe disease, include hypocalcemia, hyperglycemia, and acidosis. Hypocalcemia is most commonly caused by concomitant hypoalbuminemia. Other mechanisms include complexing of calcium to released free fatty acids, protease-induced degradation of circulating parathyroid hormone (PTH), and failure of PTH to release calcium from bone.

Acute pancreatitis is associated with a variety of local and vascular complications, including local spread of inflammation to contiguous organs. The most common include peripancreatic fluid collections, pseudocyst formation, obstruction of the duodenum or bile duct, and exocrine or endocrine insufficiency. Less common complications include pancreatic fistula formation, vascular thrombosis (i.e., splenic, portal and superior mesenteric veins), colonic necrosis, and development of an arterial pseudoaneurysm. Trypsin can activate plasminogen to plasmin and induce clot lysis. However, trypsin also can activate prothrombin and thrombin and produce thrombosis leading to disseminated intravascular coagulation.

Acute peripancreatic fluid collections are pools of peripancreatic fluid confined by normal peripancreatic fascial planes without a definable wall encapsulating the collection. These fluid collections occur during the first 4 weeks after interstitial pancreatitis. Most remain sterile and are reabsorbed spontaneously during the first several weeks after the onset of acute pancreatitis. When a localized acute peripancreatic fluid collection persists beyond 4 weeks, it is likely to develop into a pancreatic pseudocyst.

Pancreatic pseudocysts (E-Fig. 38-3) are encapsulated fluid collections with well-defined inflammatory walls and are usually located outside the pancreas with minimal or no necrosis. They occur a minimum of 4 weeks after the onset of acute pancreatitis. In symptomatic patients, if the pseudocyst is mature and encapsulated, treatment can involve endoscopic, surgical, or percutaneous drainage. Although most pseudocysts remain asymptomatic, presenting symptoms may include abdominal pain, early satiety, nausea, and vomiting due to compression of the stomach or gastric outlet. Rapidly enlarging pseudocysts may rupture, hemorrhage, obstruct the extrahepatic biliary tree, erode into surrounding structures, and become infected. Indications for pseudocyst drainage include suspicion of infection or progressive enlargement with associated symptoms previously described. Asymptomatic pseudocysts should be followed.

The term *acute necrotic collection* describes a nonorganized accumulation of heterogeneous fluid and necrotic material in the setting of necrotizing pancreatitis. The necrosis may involve the pancreatic parenchyma or peripancreatic tissue, or both. Walled-off pancreatic necrosis is a mature, encapsulated collection of pancreatic or peripancreatic necrosis that usually occurs more than 4 weeks after the onset of necrotizing pancreatitis.

Abdominal compartment syndrome is diagnosed when the intra-abdominal pressure exceeds 20 mm Hg and there are signs of new respiratory, renal, or vascular organ failure. Intra-abdominal hypertension typically occurs early and is the result of pancreatic inflammation and fluid third spacing. Abdominal compartment syndrome is associated with mortality rates ranging up to 50% to 75% in various reports. Suggested treatment includes analgesics, sedation, nasogastric tube decompression, and fluid restriction. If these measures do not result in improvement, percutaneous catheter decompression followed if unsuccessful by a surgical laparotomy is recommended. The ability of this approach to improve outcomes is the focus of ongoing research.

Diagnosis and Differential Diagnosis

The diagnosis of acute pancreatitis is based on a combination of clinical, biochemical, and radiologic factors. A diagnosis of acute pancreatitis requires two of the following three features: abdominal pain characteristic of acute pancreatitis; serum amylase or lipase levels, or both, at least three times the upper limit of normal; and characteristic findings of acute pancreatitis on imaging.

Elevated serum amylase levels may occur in a wide variety of other conditions, including bowel perforation, intestinal obstruction or ischemia, acute appendicitis, cholecystitis, tubo-ovarian disease, and renal failure. Serum amylase levels may be normal in patients with hypertriglyceridemia or alcohol-induced acute pancreatitis. Serum lipase is preferred because it is more sensitive and specific than serum amylase for the diagnosis of acute pancreatitis. The serum lipase level remains normal in some nonpancreatic conditions associated with an elevated serum amylase level, including macroamylasemia (i.e., formation of large molecular complexes between amylase and abnormal immunoglobulins), salivary gland disorders, and tubo-ovarian disease, but it may similarly rise in appendicitis, renal disease, and cholecystitis. The serum lipase concentration is more sensitive than that of amylase because it remains elevated longer and may be diagnostic even for patients seeking medical attention several days after symptom onset. Repeated measurements of serum pancreatic enzymes have little value in assessing clinical progress, and the magnitude of serum amylase or lipase elevation does not correlate with the severity of pancreatitis.

Contrast-enhanced computed tomography (CECT) or magnetic resonance imaging (MRI) of the pancreas should be used for patients whose diagnosis is unclear or who fail to improve within the first 48 to 72 hours after hospital admission. Imaging findings supporting acute pancreatitis include pancreatic enlargement, peripancreatic inflammatory changes, and extrapancreatic fluid collections. Imaging does not exclude the diagnosis of acute pancreatitis because the pancreas appears normal in 15% to 30% of those with mild disease. CECT is also useful for assessing disease severity based on the presence and extent of complications such as pancreatic necrosis and acute peripancreatic fluid collections. Pancreatic imaging should be performed after adequate fluid resuscitation to minimize the risk of contrast-induced nephrotoxicity.

MRI is preferred for patients with a contrast allergy and renal insufficiency because T2-weighted images without gadolinium contrast can similarly diagnose pancreatic necrosis. Early imaging (within 72 hours of symptom onset) can underestimate the existence and extent of pancreatic necrosis. Gallstone pancreatitis should be suspected in patients with transient elevation in liver function test results, particularly a serum ALT level elevated more than threefold. Transabdominal ultrasonography should be performed in all patients with acute pancreatitis when considering a diagnosis of gallstone pancreatitis.