

Fluid Collections and Pseudocysts as a Complication of Acute Pancreatitis

Jack Braha, MD^a, Scott Tenner, MD, MPH^{b,*}

KEYWORDS

- Pseudocysts • Pancreatic necrosis • Acute pancreatitis • Fluid collections
- Drainage

KEY POINTS

- A pseudocyst may occur secondary to acute pancreatitis, pancreatic trauma, or chronic pancreatitis. Pseudocysts usually contain a high concentration of pancreatic enzymes and variable amounts of tissue debris. Most are sterile.
- In the absence of a clear history of acute pancreatitis or chronic pancreatitis, a pancreatic cystic lesion will rarely be a pseudocyst.
- An acute fluid collection is fluid located in or near the pancreas that lacks a definite wall and typically occurs early in the course of acute pancreatitis.
- It is very difficult to distinguish acute fluid collections in the pancreatic parenchyma from pancreatic necrosis, as well as walled-off pancreatic necrosis from pseudocysts.
- Treatment choices of symptomatic pseudocysts include surgical, radiologic, and endoscopic drainage. No randomized prospective trials have adequately compared these methods.

THE NATURAL HISTORY OF ACUTE PANCREATITIS

Acute pancreatitis is among the most common diseases of the gastrointestinal tract, often leading to significant emotional, physical, and financial human burden. Clinicians often have difficulty managing patients with acute pancreatitis because the disease is complicated by an obscure pathogenesis, few effective remedies, and unpredictable outcome. The incidence of acute pancreatitis seems to be increasing.^{1,2} The increase in incidence is most likely related to the relative increasing body mass index and the

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^a Division of Gastroenterology, Mount Sinai Medical Center–Brooklyn, The Greater New York Endoscopy Surgical Center, 2211 Emmons Avenue, Brooklyn, NY 11235, USA; ^b State University of New York, The Greater New York Endoscopy Center, 2211 Emmons Avenue, Brooklyn, NY 11235, USA

* Corresponding author.

E-mail address: DrTenner@BrooklynGI.com

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increased prevalence of obesity. Because the population is becoming increasingly overweight, the incidence of gallstones, the most common cause of acute pancreatitis is rising. Fortunately, at the same time, the overall mortality rate from acute pancreatitis has gradually declined to approximately 3% to 5%.³ This is likely due to a better understanding of the pathogenesis and treatment of the disease.⁴

Acute pancreatitis seems to have 2 distinct stages.⁵ The first stage is related to the pathophysiology of the inflammatory cascade. This first phase usually lasts a week. During this phase, the severity of acute pancreatitis is related to organ failure secondary to the patient's systemic inflammatory response elicited by acinar cell injury. Fluid extravasates to the peripancreatic regions and is often referred to as acute fluid collections. Infectious complications are uncommon at this time. The fever, tachycardia, hypotension, respiratory distress, and leukocytosis are typically related to the activation of the systemic inflammatory response syndrome. Multiple cytokines are involved, including platelet-activating factor, tumor necrosis factor, and other interleukins.⁴

During the first week, the initial state of inflammation evolves dynamically with variable degrees of pancreatic and peripancreatic ischemia and/or edema, to either resolution or to irreversible necrosis and liquefaction, and/or the development of fluid collections in and around the pancreas. The extent of the pancreatic and peripancreatic changes is usually proportional to the severity of organ failure. However, organ failure may develop independent of pancreatic necrosis.⁵

Approximately 75% to 80% of patients with acute pancreatitis have a resolution of the disease process (interstitial pancreatitis) and do not enter the second phase. However, in one-quarter of patients, a more protracted course develops, typically associated with organ failure, often related to the necrotizing process (necrotizing pancreatitis), lasting weeks to months. The mortality peak in the second phase is related to a combination of factors, including organ failure secondary to sterile necrosis, or infected necrosis or complications from surgical intervention.

Similar to the 2 phases of the disease process, there are 2 peaks for mortality. Most studies in the United States and Europe reveal that about one-half the deaths occur within the first or second week of the disease, usually of multiorgan failure.⁶ Death can be very rapid. About one-quarter of all deaths in Scotland occur within 24 hours of admission and one-third within 48 hours.⁷ After the second week of illness, patients succumb to pancreatic infection associated with multiorgan failure. Some studies in Europe report a very high rate of late mortality from infection. Patients who are older and have comorbid illnesses have a substantially higher rate of mortality than younger healthier patients. In those who survive their illness, severe pancreatic necrosis can scar the pancreas, resulting in a stricture of the main pancreatic duct with subsequent recurrent attacks of disease, chronic pancreatitis, and/or recurrent pseudocyst formation.

DEFINITIONS

Acute pancreatitis is best defined clinically as the diagnosis for a patient presenting with 2 of the following criteria: (1) symptoms, such as epigastric pain, consistent with the disease; (2) a serum amylase and/or lipase greater than 3 times the upper limit of normal; or (3) radiologic imaging consistent with the diagnosis via computed tomography (CT), and/or MRI.³ Although the diagnosis of acute pancreatitis is easily determined early in the course of the disease, the severity of the disease requires vigilance because the severity of the disease is not accurately defined for the first 48 hours.⁵

After the diagnosis is established, within 48 hours of the disease, patients are classified as having severe or moderately severe or mild disease.⁵ Mild acute pancreatitis

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