Classification and Diagnosis of Acute Pancreatitis

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Received: April 2022 Accepted: May 2022; Published: June 1, 2022.
Citation: Thamin Alassaad, Shaza Youssef. Classification and Diagnosis of Acute Pancreatitis. World Family Medicine. 2022; 20(6): 78- 83. DOI: 10.5742/MEWFM.2022.9525074

Abstract

In many countries, acute pancreatitis, an inflammatory condition of the pancreas, is the main cause of gastrointestinal hospitalization. Gallstones and alcohol abuse are well-known risk factors, but several new instances have surfaced, along with new features of pathophysiology, that have improved our understanding of the disorder. The necessity for adequate therapy grows as the incidence (and admission rates) of acute pancreatitis rises. We review how to treat acute pancreatitis patients, including diagnosis, differential diagnosis, complications, prognostic factors, treatment, and prevention of second episodes, and the possibility of transitioning from acute to chronic pancreatitis.

Keywords: Pancreatic illness; stomach pain; acute pancreatitis; clinical management.

Introduction

Acute pancreatitis is a prevalent condition with a high mortality rate [1] that is typically caused by gallstone disease or excessive alcohol consumption. The presence of elevated serum amylase and lipase levels supports the diagnosis of acute pancreatitis. The amylase level rises within hours of the onset of pain and can stay there for three to five days. Although serum lipase is more selective for pancreatic disease, it can also be increased in other diseases. Hyperglycemia, hypocalcemia, leukocytosis, and moderate increases in liver function test values are also common laboratory abnormalities in acute pancreatitis. Ultrasound and magnetic resonance cholangiopancreatography may be useful in the diagnosis of acute pancreatitis, as they can detect stones in the common bile duct and assess the pancreatic parenchyma directly.

The classification, treatment, and prognosis of acute pancreatitis, as well as therapy options for acute pancreatitis complications, are discussed in the following review.

Classification of Acute Pancreatitis

Fitz made the first attempt to define the severity of acute pancreatitis in 1889, and morphological factors have always been included until the most recent Atlanta symposium in 1992 [3]. Whereas Fitz considered that signs of pancreatic bleeding and diffused fat necrosis were morphological indications of severe disease, the original Atlanta classification listed pancreatic necrosis, abscess, and pseudocyst as morphological features of severe disease [4].

According to Petrov [4], an ongoing effort is underway to modify the 1992 Atlanta categorization of acute pancreatitis in light of new findings. Persistent organ failure predicts death in acute pancreatitis, according to clinicians. Local problems without organ failure are linked to morbidity and a longer stay in the hospital, but low death. However, one of the most important aspects of the classification is the severity of acute pancreatitis [4].

A three-category classification of acute pancreatitis severity was recently created. According to Hammel et al, three new categories of acute pancreatitis severity identify patients with high morbidity and mortality (severe acute pancreatitis), high morbidity without mortality (moderate acute pancreatitis), and low morbidity without mortality (mild acute pancreatitis). www.najms.org 212 pancreatitis), North American Journal of Medical Sciences, Volume 2, No. 5, May 2010 [5]. Local consequences without organ failure characterize the condition of moderately severe acute pancreatitis. In moderately severe acute pancreatitis, mortality is similar to mild pancreatitis, but hospitalization is longer, as in severe pancreatitis. Pancreatic necrosis or fluid accumulation are examples of local problems [6].
Acute pancreatitis is divided into the following:

- Mild acute pancreatitis is defined by the absence of organ failure and local or systemic consequences.
- Moderately severe acute pancreatitis with temporary organ failure (resolves in 48 hours) and/or local or systemic consequences but no persistent organ failure (>48 hours).
- Severe acute pancreatitis is marked by persistent organ failure affecting one or more organs.

Histopathology of Acute Pancreatitis

Infected necrosis, pancreatic abscess, and/or infected pseudocysts are all possible symptoms of pancreatic infection [7]. Gram-negative bacteria such as E. coli, Enterococcus, and Klebsiella are the most common microorganisms involved [8]. Kovalska and coworkers [9] analyzed intraoperative pancreatic tissue samples for the first histological abnormalities of acute pancreatitis and attempted to uncover the possibility of pancreatic tissue restoration in the immediate aftermath of severe acute pancreatitis. Their research revealed that the exocrine portion of the pancreas suffers the most damage. Nerves and stroma appeared to be immune to the effects of pancreatitis. The stromal structure imposed some restrictions on the inflammation's progression. The resistance to inflammation of pancreatic intralobular ducts has been linked to their diameter. Inflammation is likewise rather stable on the Langerhans islands [9].

Because it is difficult to distinguish between infected and sterile pancreatic necrosis in clinical practice, needle aspiration may be required [8, 10].

Pathophysiology of pancreatic acinar cell in pancreatitis

Gallstones - Gallstones (including microlithiasis) are the most common cause of acute pancreatitis, accounting for 40 to 70% of cases. Two possible initiating events in gallstone pancreatitis have been proposed: bile reflux into the pancreatic duct due to transient obstruction of the ampulla during gallstone passage; or obstruction at the ampulla secondary to stone(s) or edema resulting from the stone passage. Recurrence is prevented by cholecystectomy and clearing the common bile duct of stones, proving the cause-and-effect relationship.

Pancreatic duct obstruction rapidly changes the physiological response of the exocrine pancreas to a Ca2+-signaling pattern that has been associated with premature digestive enzyme activation and the onset of pancreatitis [11]. Pancreatitis begins with Ca2+ signals produced by stimulation of cholecystokinin (CCK) and acetylcholine activation. CCK is known to excite mitochondria as well. A single local cytosolic Ca2+ spike lasts just a brief time, and pathological situations leading to inflammation are most likely caused by a protracted rise in the spikes. Toxic CCK causes prolonged calcium increases, which leads to the creation of post-exocytic endocytic vacuoles. In these vacuoles, trypsin is activated.

Alcohol - In the United States, alcohol is responsible for 25 to 35 percent of cases of acute pancreatitis. Approximately 10% of people with persistent alcoholism experience clinically acute pancreatitis that is indistinguishable from other types of acute pancreatitis.

Alcohol activates intracellular trypsinogen in the apical granular area in a Ca2+-dependent manner via non-oxidative metabolites such as fatty acid ethyl esters and fatty acids [12]. Intracellular trypsinogen activation is a crucial initiating event in the development of acute pancreatitis, but the specific organelle in which this process takes place has been unknown [13]. Ca2+-dependent trypsinogen activation occurs in postexocytotic endocytic vacuoles, according to new research [12]. Due to a bafilomycin-sensitive vacuolar H+ ATPase, these vacuoles are acidic and have a highly Ca2+ permeable membrane. Acid endocytic complexes, together with lysosomes, zymogen granules, and endoplasmic reticulum elements, play a key role in the physiological Ca2+ signaling that controls enzyme and fluid secretion from the exocrine pancreas [12].

Acinar cell vacuole formation and trypsinogen activation are two major pathologic acinar cell responses in acute pancreatitis [14]. The activation of trypsinogen is triggered by a rise in intracellular calcium, which is induced by the co-localization of enzymes (cathepsin B). Necrosis, apoptosis, and autophagy are the three ways to die. There is relatively little active trypsin without autophagy, but there is a lot of cytosolic cathepsin B as part of the caspase activation with autophagy. Caerulein stimulation activates caspase 3 in permeable rat acinar cells, exactly as exogenous cathepsin B activates caspase 3 [15]. Heat shock proteins, on the other hand, protect against caerulein-induced pancreatitis and have anti-cancer properties. Heat shock proteins are chaperone proteins that protect living cells against injury-inducing stimuli [16].

Hypertriglyceridemia - Acute pancreatitis can be triggered by serum triglyceride levels exceeding 1000 mg/dL (11 mmol/L).

Post-endoscopic retrograde cholangiopancreatography (ERCP) -
Acute pancreatitis affects roughly 3% of patients who have diagnostic ERCP, 5% of those who have therapeutic ERCP, and up to 25% of those who have sphincter of Oddi manometric tests. The risk of post-ERCP pancreatitis is increased by a number of operators, patient, and procedure-related variables. Lack of ERCP experience, sphincter of Oddi dysfunction, problematic cannulation, and treatment performance are all important risk factors.

Medications — Medication-induced pancreatitis is uncommon. Drug-induced pancreatitis has a usually good prognosis and a low fatality rate.

Mechanisms of drug-induced pancreatitis include immunologic reactions (eg, 6-mercaptopurine, aminosalicylates, sulfonamides), direct toxic effects (eg, diuretics, sulfonamides), accumulation of a toxic metabolite (eg, valproic acid, pentamidine, tetracycline), ischemia (diuretics, azathioprine), intravascular thrombosis (eg, estrogen), and increased viscosity of pancreatic juice (eg, diuretics and steroids) [17,18].

Imaging

Several features may be seen on imaging in patients with acute pancreatitis.

Abdominal and chest radiographs — In acute pancreatitis, radiographic findings range from unimpressive in mild disease to localized ileus of a small intestinal segment (sentinel loop) or the colon cutoff sign in more severe disease. The colon cut-off sign indicates a lack of air in the colon distal to the splenic flexure due to descending colon functional spasm caused by pancreatic inflammation. An acute peripancreatic fluid accumulation with a ground-glass appearance could be present.

On a chest roentgenogram, around one-third of patients with acute pancreatitis had anomalies such as hemidiaphragm elevation, pleural effusions, basal atelectasis, pulmonary infiltrates, or acute respiratory distress syndrome [19].

Abdominal ultrasound — On abdominal ultrasonography, the pancreas appears diffusely enlarged and hypoechoic in patients with acute pancreatitis. Gallstones in the gallbladder or bile duct can be seen.

On abdominal ultrasonography, the peripancreatic fluid appears as an anechoic collection. In the case of pancreatic necrosis, these collections may reveal internal echoes. However, intestinal gas due to an ileus prevents examination of the pancreatic or bile duct in about 25 to 35 percent of patients with acute pancreatitis [20]. Furthermore, ultrasonography is unable to distinguish between the extrapancreatic spread of pancreatic inflammation and pancreatic necrosis.

Abdominal computed tomography — Acute interstitial edematous pancreatitis is diagnosed by contrast-enhanced abdominal computed tomography (CT) scan findings of focal or diffuse pancreatic enlargement with heterogeneous enhancement with intravenous contrast. The lack of enhancement following intravenous contrast delivery indicates pancreatic necrosis.

A contrast-enhanced CT scan conducted three or more days after the onset of abdominal pain can reliably determine the presence and degree of pancreatic necrosis and local consequences, as well as forecast the severity of the disease.

A contrast-enhanced abdominal CT scan may occasionally reveal a common bile duct stone. Individuals with underlying pancreatic cancer may have a pancreatic mass, while patients with intraductal papillary mucinous neoplasia or cystic neoplasm may have diffuse dilatation of the pancreatic duct or a cystic lesion.

Magnetic Resonance Imaging — In individuals with acute pancreatitis, widespread or focal enlargement of the pancreatic gland can be detected on MR T1 weighted images with fat suppression, and the pancreatic borders may be obscured. The signal intensity of the pancreatic parenchyma may be hypointense on T1-weighted imaging and hyperintense on T2-weighted images due to pancreatic edema. Failure of the pancreatic parenchyma to enhance contrast-enhanced Magnetic Resonance Imaging (MRI) indicates the existence of pancreatic necrosis. When compared to a contrast-enhanced abdominal CT scan, MRI has a higher sensitivity for diagnosing early acute pancreatitis and can better describe the pancreatic and bile ducts, as well as acute pancreatitis sequelae [21-22]. A cholangiopancreatogram performed using magnetic resonance imaging MRCP for the identification of choledocholithiasis, is comparable to an endoscopic retrograde cholangiopancreatogram (ERCP) [23]. In comparison to iodinated contrast, MRI does not require radiation, and gadolinium has a decreased risk of nephrotoxicity [21,24,25]. A non-enhanced MRI can also detect pancreatic necrosis in patients with renal failure. After intravenous secretin, secretin MRCP (s-MRCP) is conducted, and the increased pancreatic juice induces transitory dilatation of the pancreatic duct by 1 mm or greater. It indicates a dynamic obstruction at the ampulla if it lasts longer than 5 to 10 minutes, and such patients may benefit from pancreatic sphincterotomy. The amount of opacification in the duodenum may indicate the amount of pancreatic juice released. Showing large side branches can reveal subtle underlying chronic pancreatitis. Acute pancreatitis caused by secretin is a very unusual occurrence.

However, MRI has the drawback of being operator-dependent, resulting in inconsistency in quality and technique, and its usage is constrained by the availability of local expertise. Furthermore, when compared to CT scan, MRI takes longer to scan, making it more challenging to administer in critically ill patients.

Laboratory studies

A threefold or larger increase in serum lipase or amylase over the upper limit normally indicates acute pancreatitis. We just look at serum lipase levels. When compared to amylase, lipase stays elevated for a longer duration and has a better specificity. While the patient is in the hospital, repeating pancreatic enzymes every day has no effect on management and just increases costs.

To identify the reason and rule out other causes of acute abdominal discomfort, a complete blood count, electrolytes, alanine aminotransferase (ALT), and aspartate aminotransferase (AST), bilirubin, calcium, triglyceride, and albumin should be obtained. All women of reproductive age should have a pregnancy test done.

Possible strategies for early treatment of acute pancreatitis

A genetic investigation found that toll-like receptor-4 polymorphisms may influence the likelihood of infection in acute pancreatitis [26]. In a model of chronic pancreatitis, researchers discovered that specific brain receptors, transient receptor potential vanilloid subtype 1, mediate pain responses [27].

In the acinar cell, the pancreatic zymogen chymotrypsin C can destroy pathologically activated trypsin. Inactivating mutations in chymotrypsin C have been linked to the development of chronic pancreatitis, particularly in people who abuse alcohol [27]. Not only have the enzymes changed in the early stages of inflammation, but there have also been certain unique characteristics, such as increased permeability (local and/or systemic), neuronal activation, decreased blood flow, and an acidic environment.

Acute pancreatitis is a disease that affects numerous organs, including the cardiovascular system, lungs, kidneys, pancreatic necrosis or infarction, and the gastrointestinal tract (bacterial translocation). Vascular leak syndrome is common in patients with severe acute pancreatitis, resulting in hemoconcentration, hypotension, pulmonary edema, and renal failure. Angiopoietin-1 and 2 are autocrine peptides that regulate endothelial permeability by reducing or increasing it. Serum angiopoietin-2 levels were found to be highly linked to severe acute pancreatitis and prolonged organ failure. Angiopoietin-2 levels at admission accurately predict organ failure [28].

Gallstone disease is still a common cause of acute pancreatitis in the clinic. Cholecystectomy is suggested after an episode of gallstone pancreatitis and after two occurrences of idiopathic acute pancreatitis to prevent recurrence of acute pancreatitis. In acute pancreatitis without gallbladder stones or sludge, however, cholecystectomy fails to prevent recurrence. These findings contradict the prescription for cholecystectomy in the treatment of idiopathic pancreatitis [29].

Management of pancreatic necrosis

Without drainage, sterile pancreatic necrosis is usually treated conservatively. Because it can be difficult to tell the difference between sterile and infected pancreatic necrosis, several studies have advised using CT-guided fine-needle aspiration to detect infected pancreatic necrosis. Fine-needle aspiration, on the other hand, carries significant hazards, and patients with severe pancreatic necrosis and low C-reactive protein should be closely monitored. Only when there is a clinical suspicion of infection and CRP >55 mg/L is CT-guided fine-needle aspiration recommended [30].

Debridement of severe acute pancreatitis may be timed to allow for adequate delineation of viable tissues, making debridement easier and safer. However, severe organ failure during the first week after an attack is associated with infection, making delaying treatment risky.

Open or laparoscopic surgery was used for surgical intervention. A small number of particular local problems were associated with early surgery, including hemorrhage, intestinal fistulas, and pancreatic fistulas [31]. In one study, the results of standard necrosectomy were compared to those of less invasive techniques. The majority of the 2,571 patients were handled conservatively, with only 19% undergoing surgery. Traditional necrosectomy was performed on 183 individuals (38%). Without "open" surgery, sepsis was resolved in 162 (33 percent) of the patients with organized fluid accumulation. Percutaneous puncture with laparoscopy or the extraperitoneal translumbar technique were used to implant large-bore catheters. The hospital and postoperative death rates for patients who had traditional open surgery were 6 percent and 33 percent, respectively, and 5 percent and 24 percent in the drainage group [32].

In acute pancreatitis, there are two peaks of lethality: the first occurs within the first 7-8 days after the onset of the disease and is associated with early organ failure; the second occurs in the second week and is associated with infected necrosis centers and liquid clumps [33].

A thicker wall lacking epithelial life between the necrosis and the neighboring surviving tissue creates a walled-off necrosis in necrotizing pancreatitis. Implanting two 10F drains with a nasocysticus drain for continuous lavage is advised for decompression of this fluid collection. The most prevalent issue is dirt obstructing the drains. Lavage to remove debris and contaminated materials is therefore an option, but comprehensive supplemental therapy with tailored antibiotics and jejunal feeding is required [34].
Inflammatory exudates from pancreatitis can quickly reach the spleen. Computer tomography or ultrasonography were used to assess clinical and radiological changes in the spleen. Infarct, subcapsular fluid collections, subcapsular hematoma, and abscess are all types of spleen injury. Because the spleen alterations in acute pancreatitis are transient, most patients can benefit from primary conservative therapy [35].

Conclusion

Choledocholithiasis or excessive alcohol use are major causes of acute pancreatitis. A three-category classification of acute pancreatitis severity was recently developed. A multidisciplinary approach should be used for the therapy of patients with acute pancreatitis because it is frequently difficult to manage.

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