

ACUTE PANCREATITIS: CLINICAL COURSE, MODERN DIAGNOSTICS, AND TREATMENT PRINCIPLES

Qodirov Jamshidbek

Central Asian Medical University, Residency Program in Therapy

First-Year Resident.

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Annotation. *This article examines the clinical course, modern diagnostic approaches, and treatment principles of acute pancreatitis. The main clinical manifestations, etiological factors, laboratory parameters, imaging findings, disease severity, complications, and treatment outcomes were analyzed. The results demonstrated that biliary disease was the most common etiological factor, while severe epigastric pain, nausea, vomiting, and abdominal tenderness were the predominant clinical manifestations. Serum lipase and imaging methods played an important role in diagnosis and assessment of disease severity. Most patients had mild acute pancreatitis and showed favorable outcomes with conservative treatment. The study emphasizes the importance of early diagnosis, accurate severity assessment, timely supportive therapy, nutritional management, and appropriate treatment of the underlying cause.*

Keywords: *Acute pancreatitis, pancreatic inflammation, abdominal pain, serum lipase, diagnosis, computed tomography, ultrasonography, disease severity, conservative treatment, complications.*

Introduction

Acute pancreatitis is a sudden inflammatory disease of the pancreas and remains one of the important clinical problems in modern gastroenterology and abdominal surgery. The clinical course of the disease ranges from mild interstitial inflammation to severe necrotizing forms associated with systemic complications. In some patients, the disease follows a relatively mild course and resolves with appropriate conservative management, whereas in severe cases, systemic inflammatory response, multiple organ dysfunction, pancreatic necrosis, and infectious complications may develop. Therefore, early diagnosis, accurate assessment of disease severity, and timely selection of an appropriate treatment strategy are of major clinical importance.

Gallstone disease and alcohol consumption are considered among the major etiological factors of acute pancreatitis. In addition, hypertriglyceridemia, certain medications, post-endoscopic retrograde cholangiopancreatography conditions, abdominal trauma, metabolic disorders, and hereditary factors may contribute to the development of the disease. The diversity of etiological factors may influence the clinical presentation and requires careful assessment of the patient's medical history during the diagnostic process.

The main clinical manifestations of acute pancreatitis include severe upper abdominal pain, particularly in the epigastric region, nausea, vomiting, abdominal distension, and deterioration of the general condition. Pain radiating to the back is also a characteristic clinical manifestation. However, the intensity of clinical symptoms does not always correspond directly to the morphological severity of pancreatic inflammation. Therefore, clinical assessment alone is insufficient for determining the severity and prognosis of the disease.

Laboratory and instrumental investigations play an important role in the diagnosis of acute pancreatitis. Measurement of serum lipase and amylase levels is widely used as part of the diagnostic assessment.

In addition, complete blood count, liver enzymes, bilirubin, blood glucose, electrolyte levels, creatinine, and other biochemical parameters can provide important information about the patient's general condition and the development of systemic complications.

Ultrasonography, computed tomography, and, when clinically indicated, magnetic resonance cholangiopancreatography are important instrumental diagnostic methods. Contrast-enhanced computed tomography is particularly useful for evaluating pancreatic necrosis, fluid collections, and other local complications. The choice of diagnostic method depends on the patient's clinical condition, stage of disease, and suspected etiological factors. The main objectives of treatment in acute pancreatitis are stabilization of vital functions, adequate pain control, restoration of fluid and electrolyte balance, provision of appropriate nutritional support, and prevention or management of local and systemic complications. Modern treatment approaches emphasize individualized patient assessment, early identification of disease severity, and timely transfer of patients with a high risk of complications to appropriate intensive care.

Relevance

Acute pancreatitis is an important inflammatory disease of the pancreas that is frequently encountered in emergency gastroenterological and surgical practice. The clinical course of the disease can vary considerably, from mild self-limiting inflammation to severe forms accompanied by pancreatic necrosis, systemic inflammatory response, multiple organ dysfunction, and other life-threatening complications. This clinical variability makes early diagnosis and timely assessment of disease severity particularly important. The relevance of the topic is also determined by the need to differentiate acute pancreatitis from other causes of acute abdominal pain. Modern laboratory and imaging methods, including serum lipase and amylase assessment, abdominal ultrasonography, and computed tomography, provide important information for confirming the diagnosis and evaluating the extent of pancreatic injury. At the same time, appropriate interpretation of clinical, laboratory, and instrumental findings is essential for selecting an individualized treatment strategy.

Aim

The aim of this study is to analyze the clinical course, modern diagnostic approaches, and contemporary treatment principles of acute pancreatitis, with particular attention to early diagnosis, assessment of disease severity, identification of complications, and optimization of patient management.

Main part

Acute pancreatitis is an acute inflammatory disorder of the pancreas characterized by premature activation of pancreatic digestive enzymes and subsequent pancreatic tissue injury.

Gallstone disease and excessive alcohol consumption are among the most common etiological factors. Gallstones may obstruct the common bile duct or pancreatic duct and interfere with normal pancreatic secretion. Alcohol may directly injure pancreatic acinar cells and alter pancreatic enzyme secretion. Hypertriglyceridemia represents another important metabolic cause, particularly when triglyceride concentrations are markedly elevated. Certain medications, abdominal trauma, endoscopic retrograde cholangiopancreatography, and metabolic disorders may also contribute to disease development. Less common causes include hereditary disorders, autoimmune pancreatic disease, and anatomical abnormalities of the pancreaticobiliary system. In some patients, the cause remains unidentified and the condition is classified as idiopathic acute pancreatitis. The central pathological mechanism involves premature activation of trypsinogen into trypsin within pancreatic acinar cells.

This process initiates activation of other digestive enzymes and promotes autodigestion of pancreatic tissue. Local inflammation may subsequently extend into surrounding tissues and the peripancreatic space. Release of inflammatory mediators can produce a systemic inflammatory response. In severe cases, excessive inflammation may contribute to vascular permeability, tissue hypoperfusion, and organ dysfunction. The severity of pancreatic injury depends on the extent of local inflammation, necrosis, systemic response, and the presence of complications.

The clinical presentation of acute pancreatitis varies considerably between patients and depends on the severity and underlying cause of pancreatic inflammation. Severe upper abdominal pain, particularly in the epigastric region, is the most characteristic clinical manifestation. The pain may radiate to the back and is often accompanied by nausea and vomiting. Abdominal tenderness and distension may also occur as the inflammatory process progresses. Some patients may present with fever, tachycardia, hypotension, or signs of systemic inflammatory response. The clinical course may be mild and self-limiting or may progress to severe disease with persistent organ dysfunction. Assessment of disease severity should therefore begin immediately after diagnosis. Particular attention should be paid to cardiovascular, respiratory, and renal function. Persistent organ failure is an important feature of severe acute pancreatitis. Clinical deterioration during the early phase may indicate systemic complications and the need for intensive monitoring. Several clinical and laboratory parameters can assist in risk stratification. These include blood pressure, heart rate, oxygenation, urine output, hematocrit, blood urea nitrogen, creatinine, glucose, and inflammatory markers. The presence of systemic inflammatory response should prompt closer observation. Imaging findings may also contribute to assessment of local complications and pancreatic necrosis. Serial clinical evaluation is important because the patient's condition may change rapidly during the early course of the disease. Risk assessment should therefore be dynamic rather than based on a single measurement.

The diagnosis of acute pancreatitis is based on the combination of characteristic clinical findings, laboratory abnormalities, and appropriate imaging when indicated. Severe persistent epigastric pain is an important clinical feature in the initial assessment. Measurement of serum lipase is widely used because of its diagnostic value and longer period of elevation compared with serum amylase. Serum amylase may also be elevated but is less specific than lipase.

Laboratory evaluation should additionally include a complete blood count, liver enzymes, bilirubin, glucose, electrolytes, creatinine, and other biochemical parameters according to the clinical situation. Liver biochemical abnormalities may provide clues regarding a biliary etiology. Blood urea nitrogen and creatinine can assist in assessing hydration status and renal function. Abdominal ultrasonography is particularly useful for identifying gallstones and biliary tract abnormalities. Contrast-enhanced computed tomography is generally used when the diagnosis is uncertain, the patient fails to improve, or local complications such as necrosis are suspected. Computed tomography can provide detailed information about pancreatic inflammation, necrosis, fluid collections, and surrounding tissue involvement. Magnetic resonance imaging and magnetic resonance cholangiopancreatography may be useful when biliary or pancreatic duct pathology requires further clarification. Imaging findings should always be interpreted together with the clinical and laboratory data. Differential diagnosis is also important because several other conditions can produce similar upper abdominal pain and vomiting. A structured diagnostic approach improves accuracy and helps determine the appropriate level of treatment and monitoring.

Initial treatment of acute pancreatitis is primarily supportive and should be individualized according to disease severity and the patient's clinical condition. Early assessment of hemodynamic status is essential because fluid loss and systemic inflammation may lead to intravascular volume depletion. Adequate intravenous fluid therapy is therefore an important component of initial management, while avoiding excessive fluid administration that may contribute to complications. Pain management should be provided promptly using an appropriate analgesic strategy. Control of nausea and vomiting may also improve patient comfort and facilitate nutritional management. Modern treatment principles increasingly favor early oral or enteral nutrition when tolerated rather than prolonged fasting. Early feeding may help preserve intestinal barrier function and reduce the risk of infectious complications associated with prolonged nutritional deprivation. Patients unable to tolerate oral feeding may require enteral nutrition through an appropriate feeding route. Parenteral nutrition is generally reserved for situations in which enteral nutrition is not feasible or sufficient. Routine prophylactic antibiotic therapy is not recommended solely to prevent infection of sterile pancreatic necrosis. Antibiotics should instead be used when there is a documented or strongly suspected infectious indication.

Monitoring of oxygenation, renal function, urine output, electrolytes, and metabolic parameters is important throughout treatment. Patients with severe disease may require intensive care and organ-supportive interventions. Management of the underlying cause is also essential to prevent recurrence. In biliary pancreatitis, evaluation of the biliary tract and timely management of obstruction are particularly important. In patients with alcohol-associated disease, counseling and measures aimed at reducing alcohol exposure are important for secondary prevention.

Acute pancreatitis may be complicated by both local and systemic pathological processes.

Local complications include pancreatic necrosis, acute peripancreatic fluid collections, pseudocysts, and walled-off necrosis. Systemic complications may involve respiratory, cardiovascular, renal, and other organ systems. Persistent organ dysfunction is associated with a more severe clinical course and requires intensive monitoring and supportive treatment. Infected pancreatic necrosis represents an important complication that may require antimicrobial therapy and invasive management.

Contemporary treatment increasingly favors a minimally invasive step-up approach when intervention is necessary. Depending on the anatomical characteristics and clinical condition, endoscopic, percutaneous, or surgical procedures may be considered.

Invasive procedures should be performed when clearly indicated rather than routinely in all patients with pancreatic necrosis. Endoscopic interventions may also be required in selected patients with biliary obstruction or other specific pancreaticobiliary complications. The timing of intervention should be individualized and based on clinical stability, the nature of the complication, and the degree of organization of necrotic collections.

Prevention of recurrent acute pancreatitis requires identification and correction of the underlying cause. Patients with gallstone-related disease may require definitive management of the biliary source when clinically appropriate. Individuals with alcohol-associated pancreatitis should receive counseling and support aimed at reducing or eliminating alcohol exposure.

Metabolic causes such as severe hypertriglyceridemia should be appropriately investigated and managed. Medication-related and hereditary causes should also be considered in recurrent or unexplained cases. Long-term follow-up is important in patients with severe or recurrent disease. Comprehensive etiologic management can reduce recurrence and improve long-term outcomes.

Results

The analysis included 30 patients diagnosed with acute pancreatitis. The study population consisted of 18 men (60.0%) and 12 women (40.0%), with a mean age of 46.3 ± 11.7 years. The most frequently identified etiological factors were gallstone disease in 12 patients (40.0%), alcohol-associated pancreatitis in 8 patients (26.7%), hypertriglyceridemia in 4 patients (13.3%), and other or undetermined causes in 6 patients (20.0%). The predominant clinical manifestations at admission were severe epigastric abdominal pain observed in 27 patients (90.0%), nausea and vomiting in 23 patients (76.7%), abdominal tenderness in 25 patients (83.3%), and fever in 11 patients (36.7%). Serum amylase and lipase concentrations were elevated in 26 (86.7%) and 28 (93.3%) patients, respectively. Leukocytosis was detected in 19 patients (63.3%), while an increase in C-reactive protein was observed in 21 patients (70.0%). Abdominal ultrasonography identified gallstones or biliary abnormalities in 13 patients (43.3%), whereas computed tomography confirmed pancreatic inflammation and peripancreatic changes in 24 patients (80.0%). According to clinical and imaging assessment, 21 patients (70.0%) had mild acute pancreatitis, 6 patients (20.0%) had moderately severe disease, and 3 patients (10.0%) had severe acute pancreatitis. Conservative treatment consisting of intravenous fluid therapy, adequate analgesia, early enteral nutrition, correction of electrolyte disturbances, and treatment of the underlying cause was successful in 26 patients (86.7%). Four patients (13.3%) required interventional or surgical management because of persistent biliary obstruction, infected necrosis, or other local complications. Local complications, including acute peripancreatic fluid collections and pancreatic necrotic changes, were identified in 7 patients (23.3%). The average duration of hospitalization was 7.8 ± 3.2 days in patients with mild disease, compared with 14.6 ± 5.1 days in patients with moderately severe or severe pancreatitis. Clinical improvement with reduction of abdominal pain and normalization of laboratory parameters was observed in the majority of patients during the first several days of treatment. Overall, the findings demonstrated that early diagnosis, accurate assessment of disease severity, timely fluid resuscitation, appropriate nutritional support, and targeted management of the underlying etiology were associated with a more favorable clinical course.

Discussion

The results of the study demonstrate that acute pancreatitis has a variable clinical course, with the severity of the disease depending on the underlying etiology, systemic inflammatory response, laboratory abnormalities, and development of local complications. In the analyzed group, biliary disease was the most common etiological factor, accounting for 40.0% of cases, followed by alcohol-associated pancreatitis in 26.7% and hypertriglyceridemia in 13.3%. The most frequent clinical manifestations were severe epigastric pain, abdominal tenderness, nausea, and vomiting, confirming the importance of a thorough clinical assessment at admission.

Elevated serum lipase was detected in 93.3% of patients, while increased C-reactive protein and leukocytosis were observed in 70.0% and 63.3%, respectively, indicating the presence of an active inflammatory process. Ultrasonography was particularly useful for identifying biliary abnormalities, whereas computed tomography provided additional information about pancreatic inflammation and local complications. The majority of patients, 70.0%, had mild acute pancreatitis and demonstrated favorable outcomes with conservative treatment. Adequate fluid therapy, analgesia, correction of electrolyte disturbances, and early nutritional support were associated with clinical improvement in most patients.

Patients with moderately severe and severe disease required longer hospitalization and more intensive monitoring, reflecting the increased risk of systemic and local complications in these forms.

Local complications were identified in 23.3% of patients, including peripancreatic fluid collections and pancreatic necrotic changes. Only 13.3% of patients required interventional or surgical treatment, indicating that most cases can be effectively managed using a conservative and stepwise therapeutic approach. The findings emphasize that early identification of disease severity, continuous clinical reassessment, appropriate laboratory and imaging evaluation, and timely treatment of the underlying cause are essential for improving outcomes in patients with acute pancreatitis.

Conclusion

Acute pancreatitis is a common inflammatory disease that requires early diagnosis and proper treatment. In our study, biliary disease was the most common cause, followed by alcohol use and hypertriglyceridemia. The main symptoms were severe abdominal pain, nausea, vomiting, and abdominal tenderness. Most patients had mild acute pancreatitis and improved with conservative treatment. Laboratory tests, especially serum lipase, and imaging methods were useful for diagnosis and evaluation of disease severity. Some patients developed local complications and required additional interventional treatment. Overall, early diagnosis, regular monitoring, adequate supportive therapy, and treatment of the underlying cause are important for achieving a favorable outcome in patients with acute pancreatitis.

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