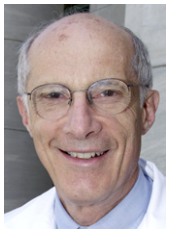
Clinical Management of Patients With Acute Pancreatitis



Bechien U. Wu[1]

Peter A. Banks[2]

[1]Center for Pancreatic Care, Southern California Permanente Medical Group, Department of Gastroenterology, Kaiser Permanente Los Angeles Medical Center, Los Angeles, California; and [2]Center for Pancreatic Disease, Division of Gastroenterology, Hepatology and Endoscopy, Brigham and Women's Hospital, Harvard Medical School, Boston, Massachusetts

Acute pancreatitis is the leading cause of hospitalization for gastrointestinal disorders in the United States. As rates of hospitalization for acute pancreatitis continue to increase, so does demand for effective management. We review approaches to best manage patients with acute pancreatitis, covering diagnosis, risk and prognostic factors, treatment, and complications, considering recommendations from current practice guidelines.

Keywords: Clinical Management; Fluid Resuscitation; Necrosis; Quality Improvement.

Acute pancreatitis is the leading cause of hospitalization for gastrointestinal disorders in the United States, with more than 280,000 hospitalizations each year.[1] The average length of stay at US hospitals in 2010 was estimated to be 5 days, at an aggregate cost of $2.9 billion. Mortality ranges from 3% for patients with interstitial (edematous) pancreatitis[2] to 15% for patients who develop necrosis.[3] As the rate of hospitalization for acute pancreatitis continues to increase,[4,5] so does the demand for effective management. This demand has resulted in publication of at least 14 clinical practice guidelines in the past decade.[6–10] An update to the American Pancreas Association and International Association of Pancreatology guidelines is forthcoming.

We review management strategies for acute pancreatitis, summarizing recommendations from current practice guidelines and discussing the latest research findings. These could help address quality improvement issues that arise in the care of patients with acute pancreatitis.

Diagnosis

Patients with acute pancreatitis have sudden onset of severe epigastric pain that occasionally radiates to their back. Accompanying symptoms frequently include nausea, vomiting, and fever or diaphoresis. Accurate diagnosis is important because many other conditions have similar symptoms, including acute cholecystitis, choledocholithiasis, and penetrating duodenal ulcers. Potentially life-threatening conditions to consider include a perforated viscus, an ischemic bowel, bowel obstruction, or myocardial infarction. The diagnosis of acute pancreatitis requires at least 2 of the following: typical upper abdominal pain, serum levels of amylase or lipase ≥3 times the upper limit of normal, and confirmatory findings from cross-sectional imaging analysis.

Disease Definitions: The Revised Atlanta Classification

The Atlanta Classification system was developed at a consensus conference in 1992 to establish standard definitions for classification of acute pancreatitis.[11] A recently completed revision of the Atlanta Classification provides a more detailed system that emphasizes disease severity and includes comprehensive definitions of pancreatic and peripancreatic collections.[12] There are also more complete definitions of local and systemic complications.

Definition of Local Complications

A variety of local complications have been delineated. Interstitial pancreatitis involves acute collection of peripancreatic fluid and formation of pancreatic pseudocysts. Necrotizing pancreatitis involves acute collection of necrosis and walled-off necrosis. Acute peripancreatic fluid collections develop during the early phase of interstitial pancreatitis. They are homogeneous in appearance without a well-defined wall, usually remain sterile, and frequently resolve spontaneously (Figure 1*A*). If an acute peripancreatic fluid collection does not resolve spontaneously, it could develop into a pseudocyst with a well-

Abbreviations used in this paper: BUN, blood urea nitrogen; CT, computed tomography; ERCP, endoscopic retrograde cholangiopancreatography; MRCP, magnetic resonance cholangiopancreatography; SIRS, systemic inflammatory response syndrome.

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defined inflammatory wall that contains fluid with very little, if any, solid material (Figure 1*B*).

An acute necrotic collection refers to the presence of necrotic tissue involving pancreatic parenchyma and peripancreatic tissues (Figure 2), only peripancreatic tissue (Figure 3), or in rare cases pancreatic parenchyma alone. These collections can be sterile or infected. If infected, they are called infected necrosis. After 4 or more weeks, an acute necrotic collection can become smaller but rarely disappears completely and usually evolves into walled-off necrosis. Walled-off necrosis has a well-defined inflammatory wall that contains varying amounts of fluid and necrotic debris (Figure 4).

Definition of Systemic Complications and Organ Failure

In the revised Atlanta Classification, systemic complications are defined as exacerbations of preexisting comorbidities such as chronic lung disease, chronic liver disease, or congestive heart failure, recognizing the failure of respiratory, cardiovascular, and renal organ systems.

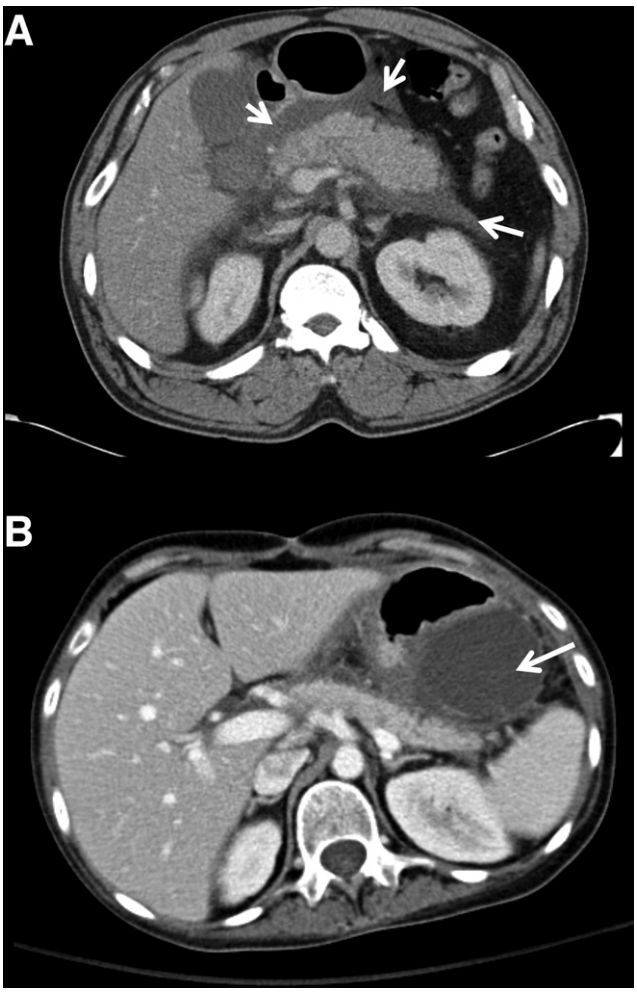

Figure 1. (*A*) Interstitial pancreatitis with acute peripancreatic fluid collection. Peripancreatic fluid collection (*arrows*) is poorly defined with homogeneous fluid density. (*B*) Resolving interstitial pancreatitis with pseudocyst. A pseudocyst (*arrow*) is typically a round or oval encapsulated collection with homogeneous fluid density.

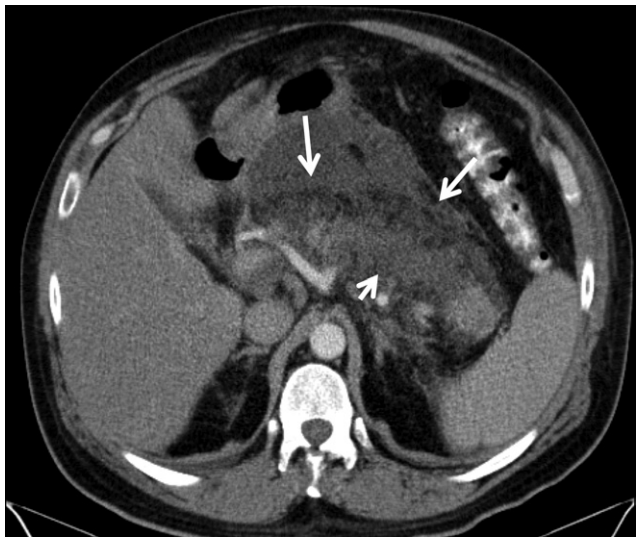

Figure 2. Pancreatic and peripancreatic necrosis. This image shows an acute necrotic collection involving both the pancreas (*large arrow*) and peripancreatic tissue (*arrowheads*).

The scoring system that has been chosen to characterize organ failure is the modified Marshall scoring system.[13] The modified Marshall system classifies disease severity on a scale from 0 to 4, so that the overall evaluation of organ dysfunction can be more completely delineated and characterized over time. In this system, organ failure is defined by a score of ≥ 2 for one or more of these organ systems.

Definition of Severity

The revised Atlanta Classification recognizes 3 degrees of severity. Mild disease is defined as acute pancreatitis not associated with organ failure, local complications, or systemic complications. Most patients with mild acute pancreatitis do not require pancreatic imaging analysis and are usually discharged within 3 to 5 days of onset of illness. Moderately severe acute pancreatitis is defined

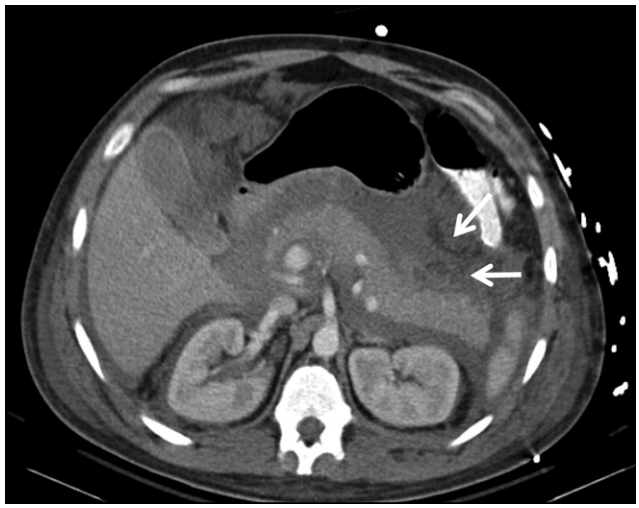

Figure 3. Acute peripancreatic necrosis is an acute necrotic collection that is heterogeneous in density. Here, the pancreas itself is inflamed (*arrows*) but not necrotic.

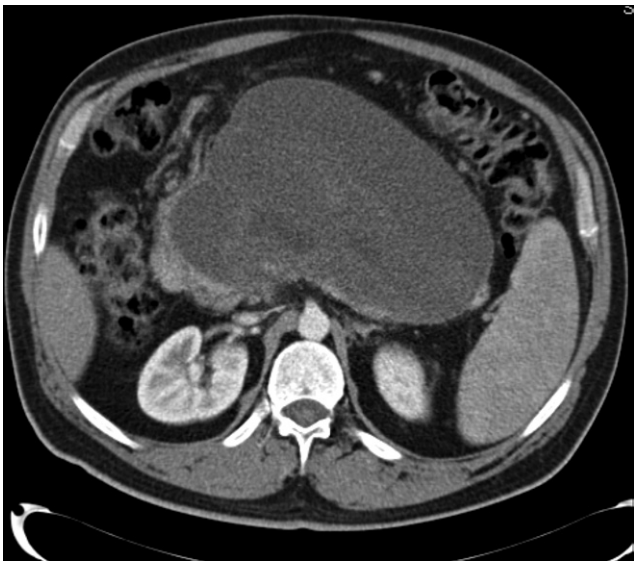

Figure 4. Walled-off pancreatic necrosis is an encapsulated collection of necrosis. This type of collection typically forms 4 to 6 weeks after disease onset. This image shows pancreatic and peripancreatic necrosis.

by the presence of transient organ failure, local complications, or systemic complications.[14] Transient organ failure is defined by organ failure that is present for <48 hours. Patients with moderately severe acute pancreatitis frequently require extended hospitalization but have lower mortality rates than patients with severe acute pancreatitis. Severe acute pancreatitis is defined by the presence of persistent organ failure. Persistent organ failure is defined by organ failure that is present for >48 hours. Most patients with persistent organ failure have pancreatic necrosis. A meta-analysis found that patients with persistent organ failure have a 30% mortality rate[15]; the risk of in-hospital death doubles when they have persistent organ failure and infected necrosis.

Roles of Advanced Imaging Techniques

The role of computed tomography (CT) in assessing patients with acute pancreatitis has changed with time.[16] A contrast-enhanced CT scan obtained within the first several days of illness cannot be used to determine whether a patient has necrotizing or severe interstitial pancreatitis. This might be because intrapancreatic fluid causes heterogeneous enhancement, which can indicate necrosis. Over a period of several days, the fluid can be reabsorbed such that a subsequent CT scan clearly shows the absence of necrosis. As such, patients should not be evaluated by CT within a few days after the onset of disease to establish the presence or extent of pancreatic necrosis.[17] The best use of an early-stage CT scan is to confirm a diagnosis of acute pancreatitis when the clinical situation is unclear. The best use of a CT scan after the first 5 to 7 days is to evaluate the presence of local complications in patients with moderately severe or severe pancreatitis to guide ongoing care.

Magnetic resonance cholangiopancreatography (MRCP) has become a useful procedure for identifying retained common bile duct stones. Selective use of MRCP can reduce the need for endoscopic retrograde cholangiopancreatography (ERCP) for patients with suspected gallstone pancreatitis.[18] Magnetic resonance imaging is helpful in distinguishing walled-off necrosis from a pseudocyst. For example, in walled-off necrosis, there are variable amounts of fluid and solid debris that can be visualized using T2-weighted imaging.

Endoscopic ultrasonography is a highly sensitive test for detecting cholelithiasis and choledocholithiasis.[19] It could be an alternative to MRCP, which has limited accuracy for detecting smaller gallstones or sludge.[20]

Risk and Prognostic Factors

It is a challenge to determine the severity of acute pancreatitis during its early stages. Extensive research has focused on risk and prognostic factors. Risk factors for severe pancreatitis include age (mortality increases among patients 60 years of age or older),[21,22] comorbid illnesses (cancer, heart failure, and chronic kidney and liver disease),[23] a history of chronic alcohol consumption,[24] and obesity (body mass index >30 kg/m^2 increases the risk of severe pancreatitis 3-fold and mortality 2-fold).[25]

The initial 12 to 24 hours of hospitalization is critical during patient management, because the highest incidence of organ dysfunction occurs during this period.[26,27] A number of clinical scoring systems and biomarkers have been developed to facilitate risk stratification during this phase. Whereas previous scoring systems such as the Ranson or Imrie–Glasgow scores required 48 hours to complete, 2 scoring systems were recently developed and involve a simplified approach that can be performed during the first 24 hours of hospitalization. The Bedside Index of Severity in Acute Pancreatitis is a 5-factor scoring system based on blood urea nitrogen (BUN) level >25 mg/dL, impaired mental status, systemic inflammatory response syndrome (SIRS), age 60 years or older, and pleural effusion (each of these criteria count as a single point).[21] A Bedside Index of Severity in Acute Pancreatitis score >2 within 24 hours is associated with a 7-fold increase in risk of organ failure and 10-fold increase in risk of mortality.[28,29]

Another scoring system, the Harmless Acute Pancreatitis Score, uses a different approach to risk stratification, identifying patients at the time of admission who are unlikely to experience complications related to acute pancreatitis.[30] Specifically, patients with a normal hematocrit and normal serum level of creatinine, without rebound tenderness or guarding, are unlikely to develop severe pancreatitis (positive predictive value of 98%). With respect to scoring systems, the most widely validated remains the Acute Physiology and Chronic Health Examination (APACHE) II score. These scoring systems have comparable levels of overall accuracy.[31]

Additional approaches have been developed to monitor disease progression. Parameters that are easy to determine

and have been validated for their ability to determine disease activity include the presence of SIRS,[32] level of BUN[27,28] or creatinine,[33] and hematocrit.[34] The presence of 2 or more of the following criteria is used to define SIRS: temperature >38.3°C or <36°C, pulse >90 beats/min, respirations >20 beats/min, and white blood cell count >12,000 or <4000 cells/mm^3 or >10% immature (bands) forms. From a clinical standpoint, tracking a patient's SIRS status offers important prognostic information; 25% to 60% of patients have SIRS when they are admitted,[35,36] but the disorder resolves in more than half of these patients within 24 hours when they are given appropriate fluid resuscitation.[35] An increasing number of SIRS criteria during the initial 24 hours of hospitalization increases the risk of persistent organ failure and necrosis as well as mortality.[36] Patients with persistent SIRS (beyond 48 hours) have 11% to 25% mortality.[36,37]

Prospective studies have shown that the level of BUN at admission and during the initial 24 hours of hospitalization is a strong prognostic factor.[38] For example, patients with a level of BUN at admission >20 mg/dL that increased during the initial 24 hours have 9% to 20% mortality. By contrast, patients with an increased level of BUN at admission that decreased at least 5 mg/dL within 24 hours have 0% to 3% mortality. A normal level of BUN at admission followed by even a modest increase (2 mg/dL) during the initial 24 hours is associated with a 6% to 15% risk of death. By contrast, patients with a normal level of BUN at admission without a subsequent increase within 24 hours have less than 1% mortality.[38,39]

A serum level of creatinine >1.8 mg/dL within the first 24 hours of hospitalization is associated with a 35-fold increased risk of development of pancreatic necrosis.[33] A persistent increase in hematocrit >44% has also been shown to increase the risk of necrosis and organ failure.[34]

Initial Resuscitation and Management

Patient management begins in the emergency department. During evaluation, the patient's diagnosis should be confirmed, risk stratification should be performed, and pain control and fluid resuscitation should be administered. Before leaving the emergency ward, patients with acute pancreatitis should be reassessed for their response to initial volume challenge and organ dysfunction.

Early Fluid Resuscitation

Aggressive volume resuscitation has been a cornerstone of therapy, based on studies in animal models and observational data from clinical studies.[40] However, approaches to fluid resuscitation require optimization.[41] Under-resuscitation during the early phase of acute pancreatitis has been associated with increased risk of necrosis and mortality.[42,43] In contrast, over-resuscitation can lead to complications such as pulmonary sequestration, as shown in recent prospective studies.[44,45]

Gastroenterologists must partner with the emergency department, hospitalist services, and critical care teams to develop institutional protocols to help ensure adequate fluid resuscitation, particularly during the initial 24 hours. Such an approach was associated with an 8.5% absolute risk reduction in mortality at a single institution over a 10-year period.[43] Studies from the sepsis literature[46] have shown that a targeted approach can improve outcome. A prospective, randomized, controlled trial assessed the effects of bolus infusion of 20 mL/kg in the emergency department, followed by continuous infusion of 3 $mL \cdot kg^{-1} \cdot h^{-1}$, with interval assessment every 6 to 8 hours (comprising vital sign monitoring, pulse oximetry, and physical examination). Repeat volume challenge was administered if the level of BUN did not decrease.[35,47] Alternatively, if the BUN level decreased, the rate of the infusion was reduced to 1.5 $mL \cdot kg^{-1} \cdot h^{-1}$. This approach was found to be safe and feasible in an acute care setting.

In general, patients undergoing volume resuscitation should have the head of the bed elevated, undergo continuous pulse oximetry, and receive supplemental oxygen. Lactated Ringer's solution reduces the incidence of SIRS by >80% compared with saline resuscitation,[35] although these findings await further confirmation. Nevertheless, lactated Ringer's solution is a reasonable choice for initial resuscitation, based on its positive effects on acid-base homeostasis, compared with large-volume saline resuscitation.[48] Because lactated Ringer's solution contains calcium, it should not be administered in quantity to patients with hypercalcemia. Volume expansion with colloid has not been shown to be more effective than with crystalloids in critically ill patients.[49]

Indications for Intensive Care

Respiratory failure is the most common form of organ dysfunction.[37] Patients with signs of respiratory failure or hypotension that fail to respond to initial resuscitation should be considered for direct admission to an intensive care unit. Patients with multiorgan dysfunction are at the greatest risk for death and should be managed in a critical care setting with a multidisciplinary care team. In addition, patients with persistent SIRS, increased levels of BUN or creatinine, increased hematocrit, or underlying cardiac or pulmonary illness should strongly be considered for management in a monitored setting.

Indications for Transfer

Data from the Nationwide Inpatient Sample indicate that patients with acute pancreatitis treated at high-volume centers (≥118 admissions/y) have a 25% lower relative risk of death than patients treated at low-volume centers.[50] Thus, patients who do not respond to initial resuscitation, with persistent organ failure or extensive local complications, should be considered for transfer to a comprehensive pancreatitis center with multidisciplinary expertise that includes therapeutic endoscopy, interventional radiology, and surgery.

Analgesia

Effective analgesia should be a priority in caring for patients with acute pancreatitis. Despite its importance, strategies to manage pain in patients with acute pancreatitis are understudied. Several practice guidelines recommend consideration of patient-controlled analgesia and administration of intermittent doses of intravenous narcotic analgesics.[51,52] There is no evidence from human studies to indicate which specific opiates are best. We recommend a comprehensive pain management approach that includes patient education, collecting patients' histories of chronic pain, and using validated pain instruments to assess pain relief.[53] Patients who receive repeated administration of narcotic analgesics should have oxygen saturation monitored.

Nutritional Support

Data from 2 randomized controlled trials support early-stage introduction of low-fat solid food as the initial meal for patients who have developed mild pancreatitis[54,55]; choledocholithiasis, duration of fasting, and quickly placing patients on a full diet have been associated with recurrence of pain.[56] For patients with more severe forms of illness or persistent abdominal pain who require further nutritional support, enteral nutrition has clear advantages over total parenteral nutrition. A Cochrane meta-analysis of 8 randomized controlled trials found a reduction in mortality, systemic infection, and multiorgan dysfunction among patients who received enteral as opposed to parenteral nutrition.[57] Several trials have proposed enteral nutrition via the nasogastric route as an alternative to nasoduodenal or nasojejunal routes.[58–60] The optimal route of enteral nutrition (nasojejunal vs nasogastric) continues to be a subject of investigation (clinicaltrials.gov NCT00580749).

Management of Local Complications

Prophylactic Antibiotics

Two high-quality, double-blind, randomized, controlled trials did not show that prophylactic antibiotics benefitted patients with necrotizing pancreatitis.[61,62] Current practice guidelines[7,9] and updated meta-analyses[63,64] did not find sufficient evidence to recommend routine use of prophylactic antibiotics in patients with acute necrotic collections (see Figure 2, for example). Overall, there has been a decrease in incidence of infected necrosis among patients even in the placebo arms of trials (15%–20% of cases with necrosis), consistent with findings from contemporary cohort studies.[65]

Necrosis

There has been a shift away from urgent surgical debridement of infected necrosis toward more conservative, less invasive approaches, indicated by the most recent international consensus for interventions in necrotizing pancreatitis.[66] In a multicenter, randomized, controlled trial from The Netherlands, a step-up approach to management of infected necrosis was compared with open necrosectomy.[67] The step-up approach involved placement of percutaneous drainage catheters in addition to treatment with antibiotics. The catheter was irrigated and upsized as necessary. Among patients whose clinical condition failed to improve within 72 hours, minimally invasive debridement was performed via a retroperitoneal approach. This step-up approach reduced major complications or death by 29% compared with traditional open necrosectomy. The median time to intervention was 29 to 30 days.

Four to 6 weeks after the onset of pancreatitis, an acute necrotic collection develops into walled-off necrosis (Figure 4). Physicians should intervene only if patients have symptoms that can be attributed to the collection (persistent abdominal pain, anorexia, nausea, or vomiting due to mechanical obstruction or secondary infection). Several studies have shown the feasibility of direct endoscopic necrosectomy using a transgastric approach for walled-off sterile necrosis.[68,69] In addition, a recent multicenter randomized trial from The Netherlands compared endoscopic with surgical necrosectomy for management of patients with walled-off infected necrosis.[70] The median time from onset of illness to intervention was 6 to 8 weeks. Endoscopic treatment reduced levels of inflammatory factors (such as interleukin-6), and the risk of new-onset multiorgan failure, intra-abdominal hemorrhage, enterocutaneous or pancreatic fistula, or death decreased by 60%. Careful patient selection and expertise with the technical aspects of direct endoscopic necrosectomy are required for successful implementation of this approach.

Pseudocyst

The incidence of pseudocysts among patients with interstitial disease appears to be less common than previously believed. A longitudinal study of patients with interstitial pancreatitis reported that most collections of acute fluid resolved within 7 to 10 days; only 6.8% of patients developed discrete pseudocysts.[71] Recent studies have indicated that symptomatic pseudocysts can be successfully decompressed by endoscopic cyst gastrostomy with endoscopic ultrasound guidance.[72]

Ductal Disruption

A ductal disruption can result in unilateral pleural effusion, pancreatic ascites, or enlarging fluid collection. Symptoms include shortness of breath, abdominal pain, and even early satiety, with vomiting if the collection compresses the stomach. Noninvasive imaging techniques such as MRCP might be used to identify a large disruption in ducts but detect small disruptions with low levels of sensitivity. ERCP is a valuable tool for treating symptomatic duct disruptions. Placement of a bridging stent across the disruption usually promotes duct healing when there is a focal disruption.[73] When a ductal disruption occurs in an area of extensive necrosis, a multidisciplinary team of therapeutic endoscopists, interventional radiologists, and surgeons should be consulted for optimal management.[74]

Peripancreatic Vascular Complications

Splenic vein thrombosis has been reported in up to 20% of patients undergoing imaging for acute pancreatitis.[75] Although gastric varices are often subsequently detected in cross-sectional image analysis, the risk of bleeding is <5%. Routine splenectomy is not recommended. Pseudoaneurysms are rare but serious complications related to acute pancreatitis, with incidences of 4% to 10%.[76] The diagnosis can be made through CT angiography. Rupture of a pseudoaneurysm can lead to life-threatening hemorrhage, with mortalities of 50% to 90%. Mesenteric angiography with transcatheter arterial embolization is considered to be the first-line treatment for pseudoaneurysms.[77]

Management of Extrapancreatic Complications

Extrapancreatic infections such as bloodstream infections, pneumonia, and urinary tract infections occur in up to 20% of patients with acute pancreatitis and increase mortality 2-fold.[78,79] If sepsis is suspected during the course of pancreatitis, it is reasonable to start antibiotic therapy while waiting for culture results. If culture results are negative, then antibiotics should be discontinued to reduce the risk of fungemia[80] or *Clostridium difficile* infection.

Comorbidities cause significant mortality among patients with interstitial or necrotizing pancreatitis. Patients should be monitored for exacerbation of underlying conditions such as congestive heart failure or chronic obstructive pulmonary disease. In addition, treatment should be provided for concurrent illnesses such as alcohol withdrawal or diabetic ketoacidosis.

Special Considerations Based on Etiology

Timing of ERCP for Patients With Biliary Pancreatitis

There are several clearly defined roles for ERCP in acute pancreatitis. Patients who have severe acute biliary pancreatitis with signs of cholangitis should undergo ERCP within 24 hours. ERCP should not be used routinely for patients with mild gallstone pancreatitis because it can increase complications.[81] Elective ERCP with sphincterotomy can be considered for patients with persistent or incipient biliary obstruction, those who are poor candidates for cholecystectomy, or those suspected of having bile duct stones after cholecystectomy.

Drug-Induced Pancreatitis

Agents reported in the literature to be associated with pancreatitis were recently reviewed.[82] Although the World Health Organization has associated more than 500 agents with acute pancreatitis, only about 30 of these have been shown to induce pancreatitis when patients were rechallenged.

Hypertriglyceridemic Acute Pancreatitis

Hypertriglyceridemia accounts for 1% to 4% of cases of acute pancreatitis.[83] Serum triglyceride levels greater than 1000 mg/dL are considered necessary to attribute an attack of pancreatitis to hypertriglyceridemia. Current first-line therapy is supportive care, as for other forms of acute pancreatitis. Case series studies have suggested use of insulin, combined with heparin or apheresis, for treatment. Administration of fibrates should begin as early as possible to help reduce the triglyceride levels. Although not as potent as fibrates, niacin or omega-3 fatty acids can be used as second-line agents.

Hypercalcemia

Acute pancreatitis with increased levels of calcium is most frequently observed in patients with hyperparathyroidism or, on occasion, metastatic tumors. It is important to treat the underlying cause of hypercalcemia to prevent recurrence of acute pancreatitis in these patients.

Autoimmune Pancreatitis

Autoimmunity is a rare cause of acute pancreatitis. Although lymphoplasmacytic sclerosing or type 1 autoimmune pancreatitis is more common, the idiopathic duct-centric type 2 form of the disease has been more frequently associated with acute pancreatitis (5% vs 34%, respectively).[84] Patients with type 2 autoimmune pancreatitis are less likely to have increased serum levels of immunoglobulin G4 or significant increases in numbers of immunoglobulin G4–positive cells based on histologic analysis. Patients with autoimmune pancreatitis are treated with glucocorticoids (typically prednisone 40 mg/day for 4 weeks, followed by a taper of 5 mg/wk). For patients who experience a relapse, treatment with immunomodulators or possibly rituximab should be considered.[85]

Prevention

Post-ERCP Pancreatitis

Placement of a pancreatic duct stent at the time of ERCP has been shown to reduce the incidence of post-ERCP pancreatitis. A meta-analysis of 8 randomized controlled trials calculated a pooled odds ratio of 0.22 for development of post-ERCP pancreatitis with stent placement.[86] Various nonsteroidal anti-inflammatory drugs have also been evaluated for their ability to prevent post-ERCP pancreatitis. Recently, a large-scale, multicenter, randomized, controlled trial showed a 45% reduction in pancreatitis when rectal indomethacin was administered immediately following ERCP to a selected high-risk population.[87]

Secondary Prevention

It is important to prevent recurrence of pancreatitis; 16.5% to 25% of patients have a repeated episode within the first several years after the initial attack.[88] Continued alcohol consumption, smoking, and recurrent

biliary complications are the major risk factors for disease recurrence. Alcohol abstinence with repeated counseling sessions at 6-month intervals is more effective than a single counseling session at reducing the frequency of recurrent acute pancreatitis.[89] Multiple societies recommend early cholecystectomy to prevent recurrent episodes of gallstone-associated pancreatitis based on early recurrence rates as high as 30% among patients awaiting cholecystectomy.[90] Among patients who are poor candidates for surgery, endoscopic sphincterotomy can reduce the likelihood of recurrent pancreatitis but is not as effective as cholecystectomy in reducing further biliary complications.[91]

Factors to Consider for Outpatients

The prevalence of exocrine insufficiency after acute pancreatitis ranges from 12% to 65%, depending on severity.[92,93] Patients with necrotizing pancreatitis, pancreatic ductal obstruction, or histories that indicate steatorrhea should receive pancreatic enzyme supplementation while they recover. Fecal elastase assays should be performed on solid stool specimens after 2 to 3 weeks to check for continued exocrine insufficiency. If exocrine insufficiency is confirmed, patients' meals should each include pancreatic enzyme extracts at doses of 40,000 to 50,000 IU lipase. The dose should be determined based on patients' symptoms, anthropometry data, and results from biochemical tests (similar to those used in treatment of patients with chronic pancreatitis).[94] In patients with extensive necrosis, significantly higher doses may be required to achieve adequate digestion.

Small cross-sectional studies have found a cumulative incidence of endocrine dysfunction of 30% to 35% among select patient populations.[92,95] Up to 30% of patients who develop endocrine dysfunction following acute pancreatitis (pancreatogenic or type 3 diabetes) require insulin therapy. Current practice guidelines do not comment on the role of screening for diabetes following acute pancreatitis. Patients with extensive necrosis or symptoms that indicate hyperglycemia should be tested for levels of hemoglobin A_{1c} or undergo glucose tolerance testing.

Several prospective studies have evaluated quality of life following acute pancreatitis. Studies using standardized quality of life instruments such as the Short Form-36 or European Organisation for Research and Treatment of Cancer survey instrument have concluded that physical function improves over time despite persistence of mental disability.[96] Long-term quality of life, even among patients who have had severe episodes of acute pancreatitis, appears to be comparable to that of the general population.[97]

Health Care Disparities

Significant racial and ethnic disparities exist in care delivery for patients with acute pancreatitis. Hispanic patients experience significantly greater delays in being seen by emergency physicians compared with white and black patients.[98] Meanwhile, black and Asian American patients with gallstone pancreatitis are significantly less likely to undergo cholecystectomy compared with white or Hispanic counterparts in the United States.[99] Further efforts are therefore needed to ensure that all patients with acute pancreatitis receive optimal care.

Quality Improvement and the Impact of Health Care Reform

Changes to the Centers for Medicare & Medicaid Services Inpatient Prospective Payment System starting in 2013 and extending into future years call for reduced reimbursement for hospital-acquired infections and hospital readmissions. These changes affect the management of patients with acute pancreatitis, who frequently develop extrapancreatic, hospital-acquired infections[78] that significantly affect mortality.[79] Also, acute pancreatitis has a high rate of recurrence; up to 20% of patients are readmitted within 30 days.[100] Risk factors for early readmission include persistent abdominal pain, inability to tolerate a full diet, hospital-acquired infection, and necrotizing pancreatitis.[101] Further efforts are clearly needed to help reduce the incidence of hospital-acquired infections and readmission for patients with acute pancreatitis.

Another important change in the shift of Medicare reimbursement to value-based purchasing will be an emphasis on patient satisfaction in acute care settings. Clinicians and researchers alike will need to adopt more patient-centric approaches that place greater emphasis on effective analgesia and preventing complications.

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Reprint requests

Address requests for reprints to: Bechien U Wu, MD, MPH, 1526 N Edgemont Ave, Los Angeles, California 90027. e-mail: bechien.u.wu@kp.org; fax: 323-783-1840

Conflicts of interest

The authors disclose no conflicts.