Acute Pancreatitis: Review and Clinical Update

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Acute pancreatitis describes an acute inflammatory process of the pancreas that can range from mild interstitial pancreatitis to severe pancreatitis with pancreatic necrosis and concomitant multiorgan failure. Acute pancreatitis is typically rapid in onset and most commonly encountered in its mild form. In 2005, more than 230,000 patients were treated for acute pancreatitis in hospitals in the United States.[1] Recent studies have demonstrated an increase in the incidence of acute pancreatitis, and some have projected substantially higher incidence rates than previously reported, although case-fatalities have remained stable since 1970.[2] The increased frequency of acute pancreatitis may be due to the rising incidence of obesity, a risk factor for the development of gallstones and, by extension, gallstone pancreatitis.[3]

Acute pancreatitis confers a heavy financial burden on the health care system and significant physiologic stress on the patient. A recent study estimated that the average cost per hospitalization for acute pancreatitis is $9870.[4] Acute pancreatitis is responsible for $2.2 billion in US health care expenditures annually. The average length of hospital stay for a patient with acute pancreatitis is approximately 5 to 6 days.[2,5,6] In addition, acute pancreatitis may be accompanied by life-threatening complications as well as significant morbidity and mortality. This article reviews the diagnosis and management of patients with acute pancreatitis.

ETIOLOGY
Gallstones and Biliary Pancreatitis

Biliary pancreatitis, synonymous with gallstone pancreatitis, is a form of acute pancreatitis caused by the passage of gallstones through the cystic duct and into the distal common bile duct where they can obstruct the biliary and pancreatic ducts. Pancreatic ductal obstruction is felt to be the inciting event in gallstone pancreatitis.[7] A study that evaluated data on consecutive patients admitted to a community hospital with pancreatitis reported that the incidence of gallstone acute pancreatitis was 45 cases per 100,000 person-

> **TAKE HOME POINTS**
>
> - Acute pancreatitis remains a commonly encountered clinical entity, with an incidence of approximately 44 cases per 100,000 adults per year.
> - The clinical diagnosis of acute pancreatitis is based on the presence of 2 of the following 3 features: serum amylase and lipase levels elevated above 3 times the upper limit of normal; mild to severe epigastric abdominal pain (often radiating to the back); and typical imaging features as found on computed tomography or magnetic resonance imaging.
> - Most patients with interstitial pancreatitis recover with conservative treatment, which may include nothing by mouth status, fluid resuscitation, and pain management, as needed. Patients with more severe acute pancreatitis require more aggressive management, and intensive care unit admission is often warranted.
> - Patients with necrotizing pancreatitis have the highest morbidity and mortality.
> - Efforts should be focused on identifying treatable causes of acute pancreatitis. If an underlying cause of acute pancreatitis is not identified, the risk of recurrence is high.

years.[7] Incidence is highest in patients with small gallstones, or microlithiasis, as these stones are more likely to escape the gallbladder and transit the cystic duct to reach the common bile duct.[8] Large stones are more likely to be retained in the gallbladder. A recent study

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noted that the incidence of all causes of acute pancreatitis rose between 1994 and 2001 from 33.2 cases to 43.8 cases per 100,000 adults, without a reduction of mortality rate. This increase was attributed to a rise in the incidence of gallstone pancreatitis.[6]

Caucasians, Hispanics, and American Indians are more likely to develop acute gallstone pancreatitis than African Americans.[6,7,9] One study demonstrated that gallstone pancreatitis typically presented at a younger age in Hispanics, who tended to have a more benign clinical course with infrequent intensive care unit (ICU) admissions.[10] Obesity is a risk factor for the development of gallstone pancreatitis and may increase the risk for the development of severe pancreatitis, including pancreatic necrosis.[11] Obesity also increases the risk of developing local complications such as pancreatic fluid collections.[11] However, obesity is not associated with increased mortality in acute pancreatitis. Pregnancy is another risk factor for the development of gallstone pancreatitis, with an incidence ranging from 1 case per 1000 to 3333 pregnancies.[12–14] Other risk factors associated with the development of gallstone acute pancreatitis include elevated alanine aminotransferase (ALT), advancing age, weight gain, female sex, and rapid weight loss.[9,15] Unsaturated fats, coffee, and moderate alcohol consumption appear to reduce the risk of developing gallstones and thus may reduce the risk for gallstone acute pancreatitis.[16–18]

Alcoholic Pancreatitis

Alcohol is a well-known precipitant of acute pancreatitis, although the incidence of acute pancreatitis in heavy alcohol consumers is not more than 2% to 3% per year, suggesting that there are as yet undetermined environmental or genetic factors that influence the development of acute pancreatitis in this population.[19] Currently, 17.6 million Americans have a form of an alcohol use disorder, and some data suggest that the incidence of alcoholic acute pancreatitis is on the rise.[5,20] Of note, the incidence rate may be similar in heavy drinkers of both sexes (91.5 cases per 100,000 persons in men versus 81.9 in women).[19] Acute pancreatitis incidence rates peak between ages 35 and 44 years.[6] Furthermore, alcoholic acute pancreatitis has the highest associated risk of overall mortality, with the odds of death increased 90% as compared with biliary pancreatitis,[6] possibly due to poor baseline nutrition.

Idiopathic Acute Pancreatitis

Between 10% and 30% of cases of acute pancreatitis may be idiopathic in nature.[21] African Americans have the highest age- and sex-standardized incidence rate of idiopathic pancreatitis.[6] A substantial proportion of acute pancreatitis of unclear cause may be explained by etiologies such as undetected microlithiasis, unrecognized drug-induced pancreatitis, or the controversial sphincter of Oddi dysfunction, among other possibilities.[22]

Inherited Forms

Mutations in the cystic fibrosis transmembrane conductance regulator (*CFTR*) allele have been associated with the development of pancreatitis. Mutations in *CFTR* can be present in as many as 10% of patients with acute pancreatitis, although it is often difficult to exclusively attribute the development of pancreatitis to *CFTR* mutations.[23] Abnormally functioning *CFTR* genes may cause inspissation of pancreatic secretions, leading to pancreatic ductal obstruction and pancreatitis.[24] *CFTR* gene mutations are often associated with recurrent acute pancreatitis and/or chronic pancreatitis. A single episode of acute pancreatitis should not prompt genetic testing for *CFTR* mutations.[25] Recurrent pancreatitis that results from *CFTR* mutations may be more aptly classified as, and often develops into, chronic pancreatitis in this population.

Hereditary pancreatitis is a rare disease characterized by mutations in the pancreatic secretory cationic trypsinogen inhibitor (*PTSI*) gene, resulting in an autosomal dominant phenotype.[26,27] Affected patients develop a form of recurrent relapsing pancreatitis that often manifests first in childhood. Hereditary pancreatitis almost always develops into chronic pancreatitis and is associated with an increased incidence of pancreatic cancer, with a cumulative incidence of pancreatic cancer of 40% by age 70 years.[28] Other rare genetic sequences, such as the MCP-1-2518 G allele, are risk factors for severity of acute pancreatitis.[29]

Drug-Induced Pancreatitis

Many drugs have been implicated as etiologic factors in acute pancreatitis, and a careful evaluation of the patient's medications is warranted in all patients with acute pancreatitis of unclear etiology. As rules of thumb, the development of acute pancreatitis must occur during treatment with the drug, all other causes of acute pancreatitis must be excluded, and the acute pancreatitis should be seen to resolve with discontinuation of the offending drug. Additionally, acute pancreatitis recurs when the drug is readministered, although this is not often tested clinically.[30] Drug-induced pancreatitis may account for as much as 7% of all cases of acute pancreatitis.[31] Medications implicated (albeit with varying levels of evidence) in the development of drug-induced pancreatitis are listed in the **Table**.[32]

Iatrogenic Pancreatitis

Iatrogenic pancreatitis most commonly occurs following endoscopic retrograde cholangiopancreatography (ERCP).[33] In a review that evaluated complication rates reported in prospective studies of ERCP, the incidence rate of iatrogenic acute pancreatitis was 3.47%, although the rate was dependent on the type of procedure performed and individual patient risk factors.[34] Studies evaluating a variety of medications to reduce the risk of post-ERCP pancreatitis have been largely unrevealing, and no currently available agent has been shown to clearly reduce the incidence of post-ERCP pancreatitis. Recent data have demonstrated that prophylactic stent placement in the pancreatic duct of high-risk patients reduces the risk of developing post-ERCP pancreatitis.[35,36] Furthermore, careful patient selection and minimally traumatic endoscopic techniques appear to offer additional protection against the development of post-ERCP pancreatitis.[33,37] Pancreatitis may also occur following abdominal surgery, cardiac surgery, liver biopsy, and abdominal procedures performed by interventional radiologists. It can also be caused by retained intra-abdominal foreign bodies as well as iatrogenic hypercalcemia due to total parental nutrition, among other causes.[38–42]

Infectious Causes

Infectious causes of acute pancreatitis are rare and have mostly been described in case reports. The most common parasitic infections linked to the development of acute pancreatitis are *Toxoplasma, Cryptosporidium,* and *Ascaris.*[43] Viruses known to cause acute pancreatitis include mumps, coxsackievirus, hepatitis B, cytomegalovirus, and varicella-zoster virus.[43] Only a few bacteria are well established as causes of acute pancreatitis: *Mycoplasma, Legionella, Leptospira,* and *Salmonella.*[44] *Aspergillus* is the only fungus that has been shown to cause acute pancreatitis.[43]

Hypertriglyceridemia and Hypercalcemia

Elevated triglyceride levels, typically exceeding 500 mg/dL, can be seen in various conditions, including poorly controlled type 2 diabetes mellitus, obesity, alcoholism, third trimester pregnancy, renal disease, hypothyroidism, and familial hypertriglyceridemia.[45,46] Patients should have fasting triglyceride levels checked after their pancreatitis has resolved before diagnosing hypertriglyceridemia, as serum triglycerides can be artificially elevated during an episode of acute pancreatitis.

Hypercalcemia is also a recognized etiology of acute pancreatitis.[44] Hypercalcemia can be associated with a malignancy (often in the setting of bony metastases

Table. Medications Implicated in the Development of Drug-Induced Pancreatitis

Class	Drug(s)
Antimicrobials	Tetracycline, sulfonamide, pentamidine, didanosine, metronidazole
Anticonvulsant	Valproic acid
Diuretic	Furosemide, thiazides
Immunosuppressant	Azathioprine, 6-mercaptopurine
Nonsteroidal anti-inflammatory	Sulindac, salicylate
Antiproliferative	Tamoxifen, L-asparaginase
Other	Estrogen

or multiple myeloma), total parenteral nutrition (as mentioned previously), sarcoidosis, vitamin D toxicity, and infusions of perioperative high-dose calcium during cardiopulmonary bypass.[47] If muscular/myopathic, urologic, or nervous system symptoms coexist with acute pancreatitis, patients should be evaluated for hyperparathyroidism.[48]

Other Causes

Less commonly encountered causes of acute pancreatitis include autoimmune pancreatitis, most often seen in the Asian population, trauma-induced pancreatitis (often following motor vehicle accidents), and scorpion stings.[49] Pediatric acute pancreatitis also occurs and can be associated with multisystem disease and systemic infection.[50] Rarely, tumors such as ampullary cancer or intraductal papillary mucinous neoplasm of the pancreas can cause acute pancreatitis. Congenital malformations, including pancreas divisum, annular pancreas, and anomalous pancreaticobiliary union, among others, have also been implicated.

RISK OF RECURRENCE

If underlying causes are not identified and treated, the risk of recurrent acute pancreatitis can be as high as 40% within 6 years. Gallstone pancreatitis carries a significantly lower risk of recurrence of approximately 10%[51] because recognition of gallstones as a cause is usually followed by a procedure to clear the bile duct and subsequent cholecystectomy. Therefore, a watch-and-wait approach cannot be advocated in these patients. A recent well-constructed meta-analysis showed a decrease in mortality following cholecystectomy in patients undergoing biliary sphincterotomy and duct clearance, further bolstering this argument.[52] Most studies report a negative correlation between mortality and recurrent acute pancreatitis.[53]

PATHOPHYSIOLOGY

Inciting events in acute pancreatitis begin in pancreatic acinar cells after a primary injury promotes pancreatic enzyme activation (primarily trypsin, although other proteases such as elastase and chymotrypsin may be involved), with subsequent enzymatic "spilling."[54] The enzymes diffuse into the interstitial and endothelial spaces and begin autodigestion of the gland. Tissue breakdown products potentiate vascular injury, with local recruitment of cytokine and arachidonic acid metabolite-secreting leukocytes. These agents produce edema and oxidative stress. The increase in vascular permeability promotes thrombosis and hemorrhage and can lead to pancreatic ischemia and necrosis.[54] Increased vascular permeability can lead to bacterial translocation into the pancreatic bed and result in infected pancreatic necrosis, a life-threatening complication of acute pancreatitis. In severe cases, systemic inflammatory response syndrome, renal failure, shock, myocardial stress, fever, or acute respiratory distress syndrome may develop.

Alcoholic acute pancreatitis may have a slightly different pathogenesis. Alcohol potently stimulates the release of secretin and cholecystokinin, which are the major contributors to pancreatic secretion.[55] Also, the rising ethanol concentration in acinar cells causes an increase in cytosolic calcium, which is required for vesicular zymogen activation. This relationship between cytosolic calcium and zymogen activation may also help to explain the association between hypercalcemia and acute pancreatitis.[55]

DIAGNOSIS

The classic presentation of acute pancreatitis includes mild to severe epigastric abdominal pain (often radiating to the back) as well as nausea and vomiting. The pain is typically constant in nature and is not aggravated by coughing, movement, or respiration. The pain tends to be more severe in a supine position and may lessen if the patient leans forward in a sitting position. Patients may appear pale and distressed, have a distended abdomen, jaundice, or tachycardia, and may be febrile.[56] Turner's sign (flank bruising) or Cullen's sign (bruising surrounding the umbilicus) may be present in severe cases. Some patients may have a more florid presentation that includes hypotension or shock due to intravascular volume depletion and third-spacing of fluids.

Commonly accepted criteria for a clinical diagnosis of acute pancreatitis necessitates the presence of 2 of the following 3 features: serum amylase and lipase levels elevated above 3 times the upper limit of normal; characteristic epigastric abdominal pain as described above; and typical imaging features as found on computed tomography (CT) or magnetic resonance imaging (MRI). Other clinical findings that can be present in acute pancreatitis include dehydration and decreased urine output. Findings that may be seen in more severe presentations include hypotension despite volume replacement and a corresponding rise in hematocrit secondary to hemoconcentration, metabolic acidosis, acute respiratory distress syndrome/respiratory failure, renal failure, and fluctuation in serum calcium levels.[57,58]

Biochemical Diagnostic Parameters

Elevation of serum amylase and lipase levels to greater than 3 times the upper limit of normal in conjunction with the appropriate clinical history are mainstays in the diagnosis of acute pancreatitis. Prospective studies comparing the selective evaluation of lipase versus amylase demonstrate a slight diagnostic advantage to lipase because amylase may have a lower sensitivity (ie, it may be normal in patients with acute pancreatitis).[59–61] In general, amylase and lipase levels do not correlate with either the severity of the attack or with overall prognosis. In addition, serum amylase and lipase levels neither assist in generating an overall prognosis nor in predicting complications of acute pancreatitis.[62–64] Most practicing physicians do not follow serum amylase and lipase levels beyond the first few days once the diagnosis has been established. A fall in enzymes accompanied by clinical improvement is often an adequate indication that the pancreatitis is resolving in most patients. Persistent elevation of serum amylase and lipase levels may suggest pancreatic ductal disruption and/or necrosis.

Serum alkaline phosphatase and bilirubin levels are not useful in isolation in the diagnosis of acute biliary pancreatitis, although a three-fold elevation of ALT has a positive predictive value of 96%, and aspartate transaminase is nearly as useful as ALT, according to one meta-analysis.[65] If liver function enzymes and amylase and lipase levels are elevated, an etiology of biliary pancreatitis is more likely, although pancreatic edema causing extrinsic compression of the distal common bile duct can produce similar laboratory findings. No single blood test has been universally shown to be of value with regards to predicting the severity of an episode of acute pancreatitis.[66] Currently, serum hematocrit and C-reactive protein (48 hr after onset) have assumed greater predictive roles in determining risk for vascular and gastrointestinal complications (*see* Prognostic Factors).[67,68]
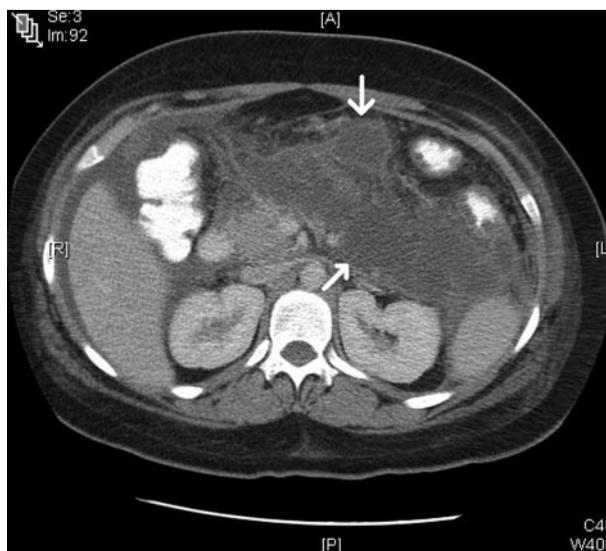
Figure 1. Computed tomography scan obtained from a patient with acute pancreatitis showing multiple well-demarcated pseudocysts approximately 6 weeks after initial presentation.

Figure 2. Computed tomography scan obtained from a patient with severe necrotizing pancreatitis (*arrows*).

Imaging

Abdominal radiographs are of some value in patients with pancreatitis. The presence of calcifications may suggest chronic pancreatitis as an underlying diagnosis. Signs that may be seen on radiograph in acute pancreatitis include a "sentinel loop," which represents a dilated segment of small intestine or colon displaying ileus, and "colon cut-off sign," which represents a functional spasm in the descending colon resulting in a termination of air in the distal colon near the splenic flexure.

A hyperechoic, diffusely enlarged pancreas is often seen on transabdominal ultrasound in acute pancreatitis. Ultrasonography is also a useful and economic choice for evaluating patients with suspected gallstone pancreatitis. This study can identify gallstones in the gallbladder, evidence of acute cholecystitis (gallbladder wall thickening or pericholecystic fluid), and common bile duct dilation (often suggestive of an obstructing common bile duct stone) and in some cases can directly visualize choledocholithiasis. Bowel gas may obscure the pancreas on ultrasound imaging.[69] Ultrasonography does not assist in diagnosing the extent of pancreatic necrosis or inflammation.

CT scans with intravenous contrast are helpful in patients with known or suspected moderate to severe pancreatitis. With this study, the entire pancreas can be well visualized and complications of pancreatitis such as fluid collections, pseudocysts (**Figure 1**), and/or areas of necrosis (**Figure 2**) can be rapidly identified. There is an excellent correlation between contrast-enhanced CT imaging results and the development of

early and late necrosis.[70] The degree of necrosis is also an excellent prognostic factor. However, small areas of necrosis can be missed via contrast CT imaging.[71]

Abdominal MRI is another commonly utilized imaging modality in patients with acute pancreatitis. Studies performed with MRI use gadolinium for contrast, which carries a lower risk of side effects or renal injury than contrast used with CT scans. MRI is also highly effective at identifying fluid collections and pancreatic necrosis.[70] It has a greater sensitivity for detecting mild acute pancreatitis as compared with CT scan.[71] MRI may be preferred over CT scan if biliary pancreatitis is suspected as magnetic resonance cholangiopancreatography can be performed at the same time and stones within the common bile duct can be readily identified.[71] Cost and accessibility limit the availability and use of MRI in rural areas.[71]

Endoscopic ultrasound (EUS) can be used to evaluate the common bile duct for the presence of stones that may require removal via ERCP. If stones are seen via EUS, ERCP can typically be performed at the same time. A meta-analysis designed to compare the accuracy of magnetic resonance cholangiopancreatography and EUS in detecting choledocholithiasis showed that the 2 modalities are equivalent in this regard.[72]

PROGNOSTIC FACTORS

Several scoring systems to assess the severity of acute pancreatitis and prognosis of patients (eg, Ranson's criteria, Imrie scoring system, and Acute Physiology and Chronic Health Evaluation II [APACHE-II]) have been
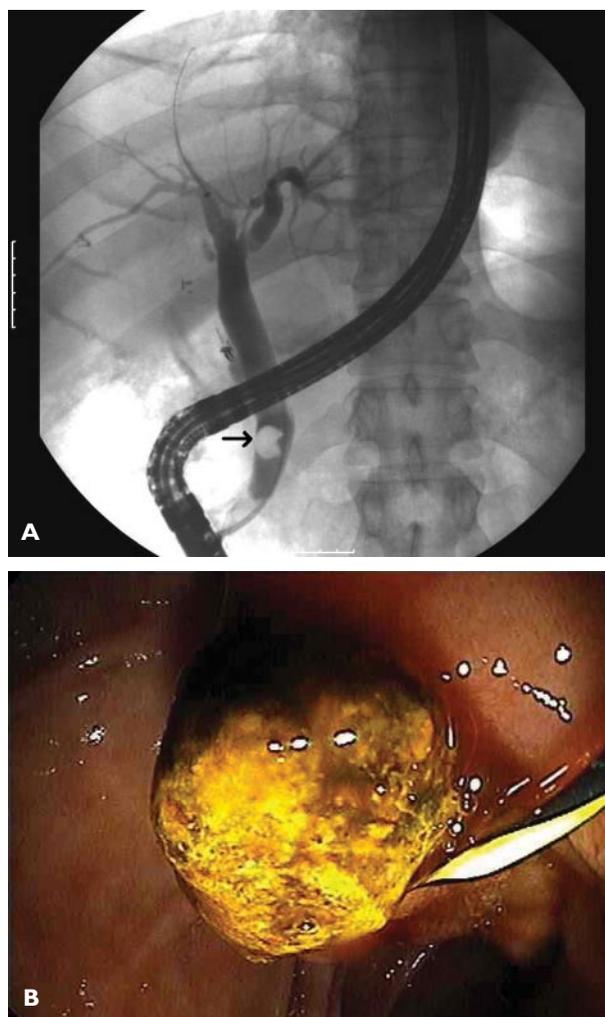
Figure 3. (**A**) Endoscopic cholangiogram obtained via endoscopic retrograde cholangiopancreatography in a patient with gallstone pancreatitis showing a 12-mm stone in the common bile duct (*arrow*). (**B**) Same stone as seen in Figure 3A following endoscopic biliary sphincterotomy and stone extraction.

developed.[73–75] In 1976, Ranson reported the use of a series of 11 objective findings that correlate with severity in patients with acute pancreatitis (eg, age, serum levels of various markers, serum calcium levels). These scoring systems may be helpful early in the clinical course of acute pancreatitis, although their usefulness is diminished as the disease progresses.[75] Generally, renal failure, respiratory failure, multiorgan system failure, fluid collections, necrosis, increased ICU length of stay, and shock are all poor prognostic factors.[76] In a study that assessed the use of scoring systems, CT, and serum markers to predict acute pancreatitis outcomes, imaging the pancreas alone was poorly predictive of outcome.[77]

Although these systems are excellent predictors of the severity of acute pancreatitis, they can be cumbersome to use. Brown and colleagues[68] have demonstrated that hemoconcentration is an early marker for organ failure and pancreatic necrosis when evaluated 24 hours after hospital admission, again suggesting the importance of adequate hydration in acute pancreatitis. Leaking exudates cause a type of third-spacing that results in a rise in hematocrit levels; this process occurs in response to cytokines released from the pancreas upon injury. Hemoconcentration markers have a sensitivity of 94% for detecting necrotizing pancreatitis and a negative predictive value of 96%.[68] Adequate hydration should be expected to reverse hemoconcentration and may reduce the risk of developing severe pancreatitis by increasing perfusion of the gland. When used as a prognostic factor, hematocrit determination is at least as accurate as APACHE-II scores but is available in half the time of the scores and is much easier to use.[78]

TREATMENT

Most patients with mild acute pancreatitis recover with supportive measures. Patients should have nothing by mouth status for at least 24 to 48 hours. In the absence of cardiopulmonary complications, vigorous hydration with intravenous fluids should be administered until adequate urine output is achieved and maintained.[68] A published study has suggested a rate of 250 to 1000 mL/hr with careful monitoring of the patient in order to establish adequate fluid resuscitation and to avoid fluid overload, although care must be individualized.[79] Pain management, typically with narcotics, should be implemented as well. Some data suggest the superiority of a patient-controlled analgesia pump with the agent meperidine instead of morphine in this setting as morphine may increase sphincter of Oddi pressure.[80] If the underlying cause of the episode of acute pancreatitis is amenable to correction (eg, choledocholithiasis), therapeutic interventions such as ERCP with biliary sphincterotomy and duct clearance and/or cholecystectomy may be indicated (**Figure 3**). Other important etiologies that may be correctable are alcohol use/abuse, hypercalcemia, hypertriglyceridemia, and drug-induced pancreatitis. There is little to be gained from daily monitoring of serum amylase and lipase levels.[59,60] Patients can be gradually returned to oral intake as abdominal pain recedes and hunger returns. Over the course of mild acute pancreatitis, most laboratory abnormalities should show improvement and resolution without further intervention within 3 to 7 days.[81]

Patients with more severe acute pancreatitis, manifested as the development of peripancreatic fluid

collection, pancreatic pseudocysts, pancreatic necrosis, and/or the development of respiratory, renal, or circulatory compromise, require more aggressive management, and ICU admission is often warranted. In general, a multidisciplinary approach with both medical and surgical teams is generally beneficial. Renal failure may warrant hemodialysis, and patients with respiratory failure may require mechanical ventilation.[82–84] Most acute fluid collections and/or pseudocysts do not require interventions unless they become infected or cause significant extrinsic compression of other organs. The management of chronic fluid collections such as pseudocysts is complex and controversial. A full discussion of the management of chronic fluid collections that result from an episode of acute pancreatitis is beyond the scope of this manuscript, but a variety of surgical, endoscopic, and percutaneous drainage procedures can be performed.

As with mild pancreatitis, nothing by mouth status is recommended in severe acute pancreatitis for at least the first 48 hours after diagnosis. If pancreatic necrosis is seen, enteral feeding using a nasoenteric tube with the distal tip placed beyond the ligament of Treitz (enteral) is often performed to reduce the risk of bacterial translocation from the gut to the necrotic pancreatic bed, improve intestinal wall integrity, and promote gut motility.[85,86] Although nasogastric tube feeding in severe acute pancreatitis appears safe according to a recent meta-analysis, an adequately powered randomized controlled study comparing it to nasojejunal feeding has been recommended and would prove useful in further investigation.[87]

Parenteral nutrition was once recommended in patients with severe pancreatitis, but it has been associated with increased length of stay, costs, and complication and mortality rates as well as increased systemic and local infections as compared with enteral nutrition.[86,88] Aggressive pain management and intravenous fluid replacement are recommended.[80,89] Vital signs and urine output should be monitored every few hours for the first 24 to 48 hours by a multidisciplinary team.

Patients who develop pancreatic necrosis must be closely monitored for the development of infected pancreatic necrosis. The risk of developing infected pancreatic necrosis rises as the proportion of involved pancreas increases.[85] The presence of physiologic organ failure may be a more important prognostic factor than the presence of sterile or infected necrosis.[90] Infected pancreatic necrosis is most commonly treated with surgical débridement, although endoscopic débridement is possible (if not widely performed), and percutaneous drainage of pus can be a consideration as well.[91] The timing of surgical intervention varies—if

patients are septic and the infected pancreatic fossa is felt to be responsible, surgical therapy may be required urgently. Delaying surgery, if possible, may allow necrotic pancreatic tissue to be well demarcated at the time of surgery.[92] Multiple surgeries may be required to fully débride necrotic pancreatic tissue.[93] Infected pancreatic necrosis, pancreatic abscesses, and infected pseudocysts are the most common indicators for surgery in the acute phase of the illness, with more minimally invasive techniques favored if possible, although open abdominal procedures may be required.[94] Patients with known or suspected infected necrosis often undergo needle aspiration to confirm infection, which can be performed via CT- or ultrasound-guided biopsy.[95,96] In rare cases, patients with infected necrosis can be treated conservatively with aggressive antibiotics and avoid or significantly delay surgical intervention.[97]

The use of prophylactic antibiotics in patients with pancreatic necrosis (in an attempt to avoid infection) remains controversial. Some studies have demonstrated benefit in this regard, while others have not shown an advantage.[98–102] Imipenem, meropenem, and fluoroquinolones are commonly used in this setting as these agents have a high degree of pancreatic penetrance. Although antibiotic prophylaxis cannot be universally recommended in the setting of acute pancreatitis, 4 situations in which administering antibiotics may be appropriate are: systemic inflammatory response syndrome or sepsis, multiorgan system failure, proven extrapancreatic or pancreatic infections, or an increase in C-reactive protein with evidence of pancreatic or extrapancreatic infection.[103]

CONCLUSION

Acute pancreatitis remains a significant cause of morbidity and mortality. Most patients with mild acute pancreatitis will recover with conservative measures, although patients who develop more severe pancreatitis, especially those with pancreatic necrosis, require aggressive management and can still have a poor outcome. A multidisciplinary approach is warranted in patients with severe pancreatitis as input from intensivists, gastroenterologists, surgeons, radiologists and other specialists is usually required. Treatable causes of pancreatitis should be identified and managed appropriately. Even with appropriate evaluation, a cause of pancreatitis may not be identified in all patients. **HP**

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