includes injection therapy (epinephrine, sclerosing agents), thermal therapy (electrocoagulation, heater probe, argon plasma coagulation), and mechanical clipping and banding.[1,15,16] All of these treatments stop bleeding, prevent recurrences, and decrease transfusion rates and length of hospital stay. The technique chosen depends on the equipment available and the experience of the endoscopist. Recently, topical therapy using a hemostatic spray and endoscopic US-guided angiotherapy has shown promising results, but additional studies are needed to confirm their utility, efficacy, and safety.[15]

Hospitalization in an intensive care setting is indicated for patients with significant upper GI bleeding due to peptic ulcers. If clinical and endoscopic features suggest a low risk of rebleeding, a ward bed may be acceptable.

PERFORATION

Perforation occurs in 2% to 14% of ulcers.[17] Its presentation is heralded by the abrupt onset of severe epigastric pain as gastric or duodenal contents spill into the peritoneal cavity, followed by the development of chemical and then bacterial peritonitis. Mortality can be as high as 10% to 40%, depending on age, comorbidities, and timeliness of diagnosis and management.[2,17] Patients may not have a history of peptic ulcer disease and may in fact have no history of ulcer-like symptoms; thus, a high index of suspicion based on history of present illness and physical exam is necessary. Elderly patients may not have dramatic pain or impressive peritoneal findings.

When the diagnosis is suspected, obtain appropriate laboratory tests, including a CBC, type and cross-match, and a lipase level; place two large-bore IV lines; provide oxygen for hypoxemia, and place a cardiac monitor; insert a nasogastric tube with suction; and obtain an acute abdominal series. Free air seen on radiographs in the setting of peritonitis on exam and appropriate history are enough to justify broad-spectrum antibiotics and prompt surgical consultation.[17] About 60% to 75% of patients with perforated peptic ulcer have free air on an upright chest radiograph.[18,19] If the plain films are ordered and do not demonstrate free air, then an abdominal CT should be ordered because the sensitivity is reported to be 98% and it can also demonstrate other potential pathology.[17-19] In some cases, nonsurgical therapy may be recommended, but operative intervention is the standard in the United States.

OBSTRUCTION

Obstruction occurs because of scarring of the gastric outlet due to chronic peptic ulcer disease, edema due to an active ulcer, or some combination of both. Resulting symptoms include abdominal fullness, nausea, and vomiting, and signs may include abdominal distention and a succussion splash. Dehydration and electrolyte imbalances may occur. Treatment includes rehydration with IV fluids, correction of electrolyte abnormalities, and relief of distention with nasogastric suction. Hospitalization is almost always indicated. The outlet may open as edema subsides, but surgical correction is often necessary.

DISPOSITION AND FOLLOW-UP

Patients with complications always require consultation, and most require admission to an appropriate inpatient unit based on the diagnosis and hemodynamic stability. Most patients with epigastric pain or dyspepsia do not leave the ED with a definitive diagnosis, but, if critical diagnoses (e.g., abdominal aortic aneurysm or myocardial ischemia) are still in the differential, obtain consultation for admission, and further evaluation is indicated. When uncomplicated peptic ulcer disease, gastritis, or dyspepsia is strongly suspected, the great majority of patients can be discharged with acid-suppressive therapy with a proton pump inhibitor or an H_2 receptor antagonist and instructions to follow up with their primary care providers. If alarm features (indicating possible cancer or bleeding) are present, obtain consultation for early endoscopy.

Discharge instructions should include an explanation of the diagnosis and home treatment, specific follow-up instructions, and warning symptoms that should prompt immediate reevaluation. The explanation of the diagnosis should specify that peptic ulcer disease is a presumptive diagnosis and that more definitive diagnostic testing may be necessary. Instructions for home treatment should include a reminder to take medications as directed; a warning against use of alcohol, tobacco products, and aspirin or other NSAIDs; and a recommendation to avoid foods that appear to upset the individual's "stomach." Specific follow-up instructions should include the name and phone number of the appropriate provider whenever possible and a time frame for reevaluation, generally 24 to 48 hours if not improving or 1 to 2 weeks if improving. Warning symptoms that merit immediate reevaluation include those that may be attributed to ulcer complications or confounding illness: worsening pain, increased vomiting, hematemesis or melena, weakness or syncope, fever, chest pain, radiation of pain to the neck or back, and shortness of breath.

REFERENCES

The complete reference list is available online at www.TintinalliEM.com.

CHAPTER 79

Pancreatitis and Cholecystitis

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PANCREATITIS

INTRODUCTION/EPIDEMIOLOGY

Pancreatitis is an inflammatory process of the pancreas that may be limited to just the pancreas, may affect surrounding tissues, or may cause remote organ system dysfunction. Most patients will have only one episode of acute pancreatitis, whereas 15% to 30% will have at least one recurrence.[1-3] Between 5% and 25% of patients will ultimately develop chronic pancreatitis.[2,3]

Most cases (~80%) involve only mild inflammation of the pancreas, a disease state with a mortality rate of <1%, which generally resolves with only supportive care.[1,4] A small proportion of patients suffer from more severe disease that may involve pancreatic necrosis, inflammation of surrounding tissues, and organ failure.[5,6]

Factors associated with acute pancreatitis are listed in **Table 79-1**.[7-11] Most cases are related to either gallstones or alcohol consumption. About 5% of all patients who undergo endoscopic retrograde cholangiopancreatography for treatment of gallstones develop pancreatitis within 30 days.[7]

Alcohol use and pancreatitis have a complex relationship, thought to be founded in toxicity and immunologic mechanisms.[12]

MEDICATIONS

Over 500 drugs have been linked to acute pancreatitis, but together, they account for fewer than 2% of cases. **Table 79-2** lists the drugs found to be most well linked to acute pancreatitis based on number of case reports and recurrence after drug reexposure.[13]

Medications associated with acute pancreatitis can be categorized into three groups: antiretrovirals, chemotherapy, and immunosuppressants. Patients taking these medications are at particular risk of severe disease because of the underlying disease combined with the medication side effects. 2′,3′-Dideoxyinosine can cause potentially fatal pancreatitis, whereas patients receiving the antiretrovirals lamivudine and nelfinavir are at lower risk.[13]

Cancer patients undergoing chemotherapy with one or more of seven medications have a risk of pancreatitis complicating the disease course.

TABLE 79-1 Causes of Acute Pancreatitis

Common	Gallstones (35%–75%)[8] Alcohol (25%–35%)[8] Idiopathic (10%–20%); increases with age[9]
Uncommon	Hypertriglyceridemia (fasting triglycerides >1000 milligrams/dL) (1%–4%)[10] Endoscopic retrograde cholangiopancreatography[7] Drugs (1.4%–2%); usually mild disease
More uncommon (total <8% of cases)	Abdominal trauma Postoperative complications, especially post–cardiopulmonary bypass Hyperparathyroidism Infection (bacterial, viral, or parasitic) Autoimmune disease Tumor (pancreatic, ampullary) Hypercalcemia Cystic fibrosis
Rare	Ischemia Posterior penetrating ulcer Toxin exposure
Unknown	Congenital abnormalities[11]

These medications are L-asparaginase, cisplatin, cytarabine, ifosfamide, mercaptopurine, pegaspargase, and tamoxifen.[13] These agents are used to treat leukemias, lymphomas, sarcomas, and breast, cervical, lung, ovarian, and testicular cancers.

Patients receiving azathioprine for posttransplantation immunosuppression or treatment of inflammatory diseases such as rheumatoid arthritis and inflammatory bowel disease are also at risk of developing pancreatitis.[13]

PATHOPHYSIOLOGY

The pathophysiology of pancreatitis is not completely understood. Under normal circumstances, trypsinogen is produced in the pancreas and secreted into the duodenum where it is converted into the protease trypsin. In acute pancreatitis, for unclear reasons, although possibly related to transient obstruction of the pancreatic duct, trypsin is activated within the pancreatic acinar cells. Activation continues in an unregulated fashion, and elimination of activated trypsin is inhibited, resulting in high pancreatic levels of activated trypsin. Activated trypsin in turn activates other digestive enzymes, complements, and kinins, leading to pancreatic autodigestion, injury, and inflammation. Pancreatic injury activates local production of inflammatory mediators, which cause further inflammation.[14,15] Fortunately, most cases never progress beyond local inflammation. However, in a minority of cases, termed necrotizing pancreatitis, pancreatic injury progresses to involve surrounding tissue or possibly remote organ systems.[16] The release of inflammatory mediators from the pancreas, in particular from the acinar cells, and extrapancreatic organs such as the liver leads to remote organ injury and failure, the systemic inflammatory response syndrome, multiorgan failure, and even death.[14-16]

TABLE 79-2 Drugs Associated With Acute Pancreatitis[13]

- Acetaminophen
- Amiodarone
- Angiotensin-converting enzyme inhibitors and angiotensin receptor blockers (enalapril and losartan)
- Antibiotics (erythromycin, metronidazole tetracycline, trimethoprim-sulfamethoxazole)
- Antiepileptics (carbamazepine, valproic acid)
- Azathioprine
- Cannabis
- Chemotherapy agents (6-mercaptopurine, cisplatin, L-asparaginase, ifosfamide, tamoxifen, cytarabine, pegaspargase)
- Codeine (and other opiates)
- Dexamethasone (and other steroids)
- Didanosine
- Diuretics (chlorothiazide, hydrochlorothiazide, furosemide)
- Estrogens
- Mesalamine
- Methimazole
- Pravastatin/simvastatin
- Tuberculosis antibiotics (dapsone, isoniazid, rifampin)

CLINICAL FEATURES

HISTORY AND PHYSICAL EXAMINATION

Acute pancreatitis causes acute, severe, and persistent abdominal pain, usually associated with nausea, vomiting, anorexia, and decreased oral intake.[17] The pain is located in the epigastrium or occasionally in one or both upper quadrants. Pain may radiate to the back, chest, or flanks. Pain may worsen with oral intake or lying supine and may improve with sitting up with the knees flexed.[18-20] Other symptoms include abdominal distention, diaphoresis, hematemesis, and shortness of breath. **Pain described as lower abdominal pain or dull or colicky pain is highly unlikely to be pancreatitis**.[19]

Vital signs may be abnormal, with tachycardia, tachypnea, fever, or hypotension. Pain is often associated with guarding and decreased bowel sounds.[17] Occasionally patients will be jaundiced, pale, or diaphoretic.

Rare physical findings associated with late, severe necrotizing pancreatitis include Cullen's sign (bluish discoloration around the umbilicus signifying hemoperitoneum), Grey-Turner sign (reddish-brown discoloration along the flanks signifying retroperitoneal blood or extravasation of pancreatic exudate), and erythematous skin nodules from focal subcutaneous fat necrosis.[20,21]

DIAGNOSIS

Formal diagnosis is based on at least two of three criteria: (1) clinical presentation consistent with acute pancreatitis, (2) a serum lipase or amylase value significantly elevated above the upper limit of normal, or (3) imaging findings characteristic of acute pancreatitis (IV contrast-enhanced CT, MRI, or transabdominal US).[19,22] The differential diagnosis is wide and consists of all causes of upper abdominal pain, as detailed in Chapter 71, "Acute Abdominal Pain."

LABORATORY STUDIES

There is no gold standard laboratory diagnosis for acute pancreatitis. Two current guidelines recommend that the amylase or lipase value be at least three times the upper limit of normal[19,22]; some recommend a lipase of two times normal or an amylase of three times normal in a patient with the appropriate clinical presentation[23]; and some recommend that any elevation above normal is consistent with the diagnosis.[17] Normal levels for amylase and lipase are based on values in young, healthy patients, making it difficult to determine applicable levels for older patients or those with multiple comorbidities.[23] Consequently, the combination of an elevated laboratory value with a clinical presentation consistent with pancreatitis is key for diagnosis.[19]

Amylase is not a good choice for diagnosis.[19] Amylase rises within a few hours after the onset of symptoms, peaks within 48 hours, and normalizes in 3 to 5 days.[23] About 20% of patients with pancreatitis, most of whom have alcohol- and hypertriglyceridemia-related disease, will have a normal amylase.[24] Because of these facts, amylase has a sensitivity of about 70%, with a positive predictive value ranging from 15% to 72%.[17] Amylase can be elevated in multiple non–pancreas-related diseases, such as renal insufficiency, salivary gland diseases, acute appendicitis,

cholecystitis, intestinal obstruction or ischemia, and gynecologic diseases, lowering its specificity for pancreatitis.[17,24]

Lipase is more specific to pancreatic injury and remains elevated for longer after the onset of symptoms than amylase. Although lipase may be elevated in diabetes and some nonpancreatic diseases such as renal disease, appendicitis, and cholecystitis, it is less associated with nonpancreatic diseases than amylase.[19,25] Lipase is more sensitive both in patients with a delayed presentation and in pancreatitis associated with alcohol use and hypertriglyceridemia.[23]

If a combination of elevated lipase and amylase is used to diagnose pancreatitis, the diagnosis is more specific and less sensitive than when using elevation in only one value; however, there is no evidence that adding amylase to a nondiagnostic lipase improves diagnostic accuracy over lipase alone.[23]

The urine trypsinogen-2 dipstick test is a rapid, noninvasive test with high sensitivity (82%) and specificity (94%).[26] However, given its current limited availability, it is not included as part of the diagnostic criteria for pancreatitis.[22]

In addition to serum lipase and amylase, obtain blood studies to evaluate renal and liver function, electrolyte status, glucose level, WBC count, and hemoglobin/hematocrit. These lab results help the clinician predict disease severity and outcome (detailed below), optimize the clinical status of the patient, identify complications that need immediate treatment (cholangitis, organ failure), and assess effectiveness of treatment.

An **alanine aminotransferase** of >150 U/L within the first 48 hours of symptoms predicts gallstone pancreatitis with a greater than 85% positive predictive value.[27]

IMAGING

Imaging can identify the cause of pancreatitis and can identify complications and severity. For patients with acute pancreatitis where gallstones have not been excluded, obtain a transabdominal US in the ED to detect gallstone pancreatitis.[3,22,28] For any patient with respiratory complaints, obtain a chest radiograph to evaluate for pleural effusions and pulmonary infiltrates, both associated with more severe pancreatitis.

In patients who meet the clinical presentation and laboratory criteria, routine early CT, with or without IV or PO contrast, is not recommended for multiple reasons. Most patients have uncomplicated disease and are readily diagnosed by clinical and laboratory criteria. There is no evidence that early CT, with or without contrast, improves clinical outcomes, possibly because CT findings are delayed compared to clinical presentation and may underestimate disease severity.[22,29,30] Peripancreatic fluid collections or pancreatic necrosis detected by CT of any kind within the first few days of symptoms generally require no treatment, and the complete extent of these local complications is usually not appreciated until at least 3 days after onset of symptoms. The magnitude of morphologic change on imaging studies does not necessarily correlate with disease severity.[31] Finally, IV contrast infusion can cause allergic reactions, nephrotoxicity, and worsening of pancreatitis.[32]

If the clinical diagnosis of acute pancreatitis is in doubt, consider further evaluation with IV contrast **abdominal CT**. Characteristic findings include: (1) pancreatic parenchymal inflammation with or without peripancreatic fat inflammation; (2) pancreatic parenchymal necrosis or peripancreatic necrosis; (3) peripancreatic fluid collection; or (4) pancreatic pseudocyst.[19,33] **Figure 79-1A–D** compares CT image of a normal pancreas to images in various complications. Although noncontrast MRI is not readily available to the ED, this imaging modality can identify the complications of pancreatitis and choledocholithiasis. It can be an alternative for patients with renal failure, patients who are allergic to IV contrast, or pregnant patients.[34]

TREATMENT

Treatment is supportive and symptom based (Table 79-3). No specific medication effectively treats acute pancreatitis; however, early aggressive hydration decreases morbidity and mortality.[35-37] The benefit of fluid resuscitation may result from increased micro- and macrocirculatory support of the pancreas, which prevents complications such as pancreatic necrosis.[38]

Provide fluid resuscitation. Fluid loss results from vomiting, third spacing, increased insensible losses, and decreased oral intake. Patients generally need a total of 2.5 to 4 L of fluid over the first 12 to 24 hours.[19,22] The specific rate of fluid delivery depends on the patient's clinical status. In the situation of renal or heart failure, deliver fluid more slowly to prevent complications such as volume overload, pulmonary edema, and abdominal compartment syndrome. Crystalloids are the resuscitation fluids of choice. Normal saline in large volumes may cause a nongap hyperchloremic acidosis and can worsen pancreatitis, possibly by activating trypsinogen and making acinar cells more susceptible to injury.[19,39] A single randomized study showed a decreased incidence of systemic inflammatory response syndrome in patients who received lactated Ringer's instead of 0.9% normal saline.[39] Regardless of which fluid is selected, monitor vital signs and urine output for response to hydration.

Control pain and nausea. Pain control is best achieved with IV opioid analgesics. Initially, place patients on NPO (nothing by mouth) status and administer antiemetics. There is no benefit to nasogastric intubation.

Prolonged bowel and pancreas rest increases gut atrophy and bacterial translocation, leading to infection and increasing morbidity and mortality.[40] In the ED, if nausea and vomiting have resolved and pain has decreased, transition the patient to oral pain medications and small amounts of food.[41] A low-fat solid foods diet provides more calories than a clear liquid diet and is safe.[42]

Acute pancreatitis by itself is not a source of infection, and prophylactic use of antibiotics and antifungals is not recommended.[43] Administer antibiotics if a source of infection is demonstrated, such as cholangitis, urinary tract infection, pneumonia, or infected pancreatic necrosis.[43]

COMPLICATIONS OF ACUTE PANCREATITIS

Although most patients with acute pancreatitis have mild uncomplicated disease, a small percentage of patients have more severe disease. In the ED, it is difficult to distinguish disease severity, because most patients present so early in the disease course that complications that define moderately severe or severe disease are not evident. Moderately severe acute pancreatitis is characterized by transient organ failure (<48 hours), local complications, or systemic complications. Severe disease includes one or more local or systemic complications and persistent organ failure (>48 hours). Critical acute pancreatitis is defined as persistent organ failure and infected pancreatic necrosis.[44]

Local complications, including acute peripancreatic fluid collections, pancreatic pseudocyst, acute pancreatic or peripancreatic necrosis, walled off necrosis, gastric outlet dysfunction, splenic and portal vein thrombosis, and colonic inflammation/necrosis, are not usually well demonstrated on CT scan until at least 72 hours after the onset of symptoms.[16] Suspect local complications in patients who have persistent or recurrent abdominal pain, an increase in pancreatic enzyme levels after an initial decrease, new or worsening organ dysfunction, or sepsis (fever, increased WBC count).

Organ failure can be seen in any system, but three organ systems are particularly susceptible: cardiovascular, respiratory, and renal. Because of the susceptibility of these three organ systems, pay special attention during the patient's initial evaluation.

Other possible complications of acute pancreatitis are listed in **Table 79-4.**

PREDICTION OF DISEASE SEVERITY

Many different scoring systems exist, including the Ranson criteria, Acute Physiology and Chronic Health Examination-II, modified Glasgow score, Bedside Index for Severity in Acute Pancreatitis, and Balthazar CT Severity Index. These scoring systems include many data points, some of which are not collected until at least 48 hours after presentation, limiting their utility in the ED.[45] None of these scoring systems is superior to another, and all have high false-positive rates.[46] **Systemic inflammatory response syndrome at admission and persistent at 48 hours predicts severe acute pancreatitis more simply and as accurately as the various scoring systems.**[6,46,47] **Besides systemic inflammatory response syndrome, a number of other clinical findings at initial assessment are associated**

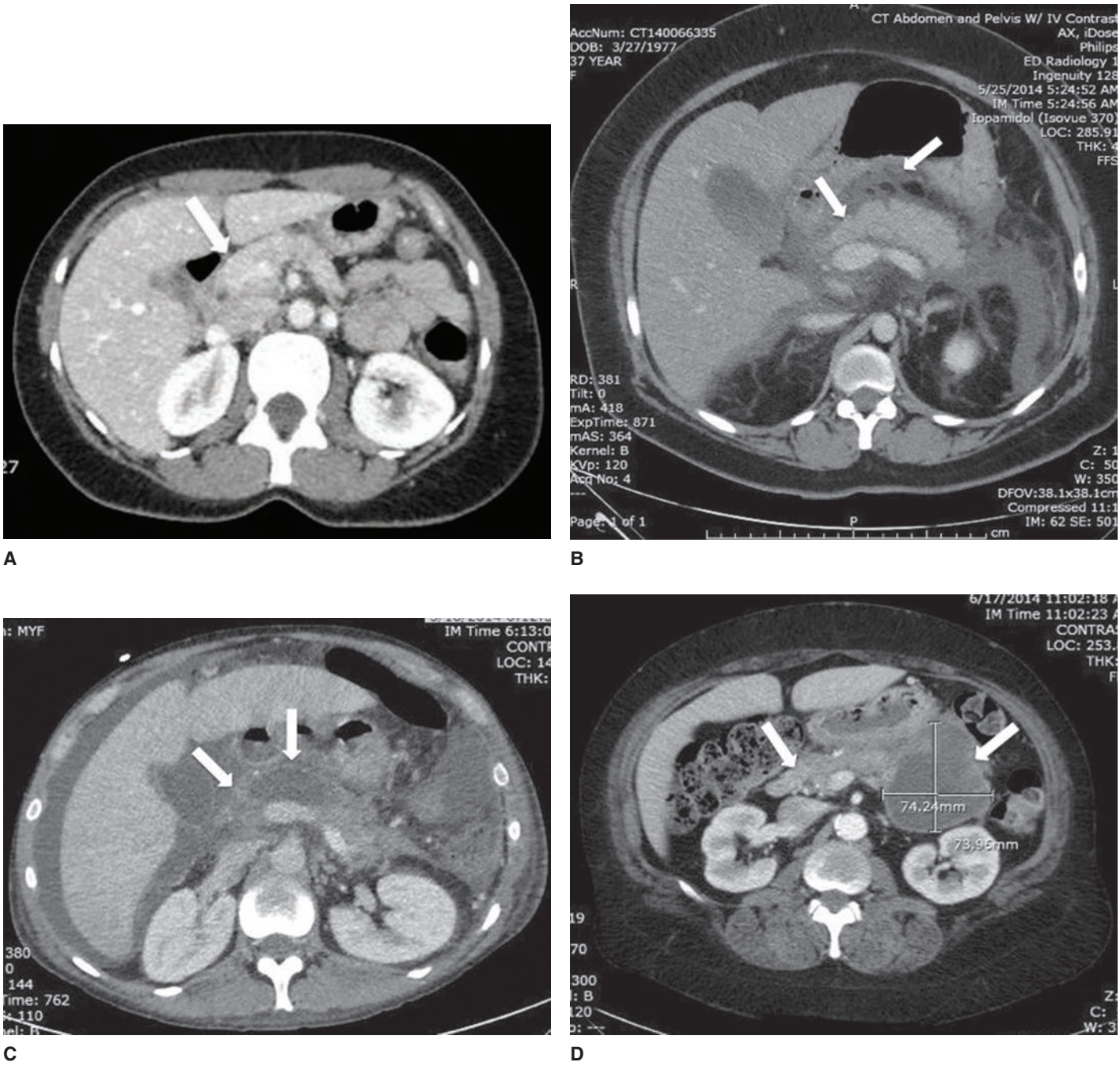

FIGURE 79-1. Abdominal IV contrast-enhanced CT scans showing: **A.** normal pancreas (*arrow*) with smooth outer contours, clear demarcation between pancreas and surrounding tissues, and without peripancreatic fluid; **B.** mild pancreatitis with indistinct pancreatic borders (*left arrow*), pancreatic edema, and peripancreatic fluid (*right arrow*); **C.** edematous pancreas with indistinct borders (*left arrow*) and area of nonenhancing parenchyma pancreatic necrosis with area of acute pancreatic necrosis (low attenuation representing nonenhancing parenchyma; *right arrow*); and **D.** edematous pancreas with indistinct pancreatic borders (*left arrow*) and a pseudocyst in the pancreatic tail (*right arrow*). [Images contributed by Bart Besinger, MD, FAAEM.]

with severe disease. These findings include patient characteristics (age >55 years, obesity, altered mental status, comorbidities), laboratory findings (BUN >20 milligrams/dL or rising; hematocrit >44% or rising; increased creatinine), and radiologic findings (many or large extrapancreatic fluid collections, pleural effusions, pulmonary infiltrates).[16,35,48]

Overall, acute pancreatitis has a mortality rate of approximately 1%.[4] Moderately severe and severe disease mortality rates are 5% and 30%, respectively.[6,49] Most patients who die do so from multiorgan failure. The sensitivity of systemic inflammatory response syndrome on admission for mortality is 100% with a specificity of 31%, whereas the sensitivity and specificity of systemic inflammatory response syndrome at 48 hours (persistent systemic inflammatory response syndrome) are 77% to 89% and 79% to 86%, respectively.[6,47] Systemic inflammatory response syndrome at admission and 48 hours, combined with patient characteristics (age, comorbidities, and obesity) and response to treatment, helps predict outcome.

DISPOSITION AND FOLLOW-UP

Patients with nonbiliary pancreatitis whose pain can be controlled in the ED and who can tolerate oral feeding can be discharged. Patients who are discharged from the ED should be referred for appropriate follow-up to help prevent recurrence.

Consider admission for a first bout of acute pancreatitis, for any case of biliary pancreatitis, and for patients needing frequent IV pain

TABLE 79-3 Treatment of Acute Pancreatitis

Treatment	Comments
Aggressive crystalloid therapy	Lactated Ringer's preferably 2.5–4 L, at least 250–500 mL/h or 5–10 mL/kg/h Use caution in congestive heart failure, renal insufficiency Monitor response: – Hematocrit 35%–44% – Maintain normal creatinine – Heart rate <120 beats/min – Mean arterial pressure 65–85 mm Hg – Urine output 0.5–1 mL/kg/h (if no renal failure)
Vital signs/pulse oximetry	Monitor closely/frequently; initially at least every 2 h, but patients may require more frequent monitoring
Electrolyte repletion	Correct low ionized calcium, hypomagnesemia Control hyperglycemia
Pain control	Parenteral narcotics
Supplemental oxygen	As needed for respiratory insufficiency
Antiemetics	Control nausea/vomiting NPO status with early transition back to oral intake Nasogastric tube/suction typically not indicated
Antibiotics	If known or strongly suspected infection, give appropriate antibiotics based on cause Not indicated prophylactically or for mild pancreatitis
Consultation for endoscopic retrograde cholangiopancreatography	In first 24 h for those with documented biliary obstruction or cholangitis

Abbreviation: NPO = nothing by mouth.

medication, not tolerating oral intake because of vomiting or increasing pain, with persistent abnormal vital signs, or with any signs of organ insufficiency (e.g., increased creatinine).

Admit to the intensive care unit a patient with severe pancreatitis or anyone who meets local criteria for an intensive care or at least an intermediate care unit admission. Biliary pancreatitis requires either admission by surgeon or early surgical consultation for consideration of early cholecystectomy.[50] Cholecystectomies in patients not suffering from documented gallstone pancreatitis are associated with increased recurrence of acute pancreatitis.[51]

Patients with cholangitis or known biliary obstruction on admission may benefit from early endoscopic retrograde cholangiopancreatography.[52] Early routine endoscopic retrograde cholangiopancreatography in patients without one of these two complications does not improve mortality or modify or prevent local complications.[52]

SPECIAL CONSIDERATIONS

CHRONIC PANCREATITIS

Chronic pancreatitis is a continuum of acute pancreatitis. From 5% to 25% of patients can progress to chronic pancreatitis.[2,3] Progression is most common in alcohol-induced disease, but may happen in any situation.[2,3]

Attacks are similar to acute pancreatitis. The goal of treatment is hydration and pain and nausea control. The mortality risk of chronic pancreatitis recurrences is generally lower than that of acute pancreatitis.[2,3]

CHOLECYSTITIS

INTRODUCTION AND EPIDEMIOLOGY

Cholecystitis is inflammation of the gallbladder that is usually caused by an obstructing gallstone.

Gallstones produce disease states, including acute cholecystitis, that vary considerably in their severity, clinical presentation, and management strategies. In the United States, the prevalence of gallstones is 8% among men and 17% among women.[53] Prevalence increases with age and with increasing body mass index. Bariatric surgery is also a risk factor for the development of gallstones.[54] The vast majority of gallstones are asymptomatic. *Asymptomatic gallstones* may be discovered incidentally on diagnostic imaging. The risk of developing symptoms or complications is 1% to 4% per year.[55]

Biliary colic is the most common complication of gallstone disease. Patients experience recurrent attacks of upper abdominal pain that typically last no more than a few hours and resolve spontaneously when the gallstone moves from its obstructing position. If the obstructing stone remains in place, acute cholecystitis may develop over time as the gallbladder becomes distended, inflamed, and in some cases infected. As acute cholecystitis evolves, it may result in necrosis and gangrene of the gallbladder wall (**gangrenous cholecystitis**). **Emphysematous cholecystitis** occurs when the inflamed gallbladder becomes infected with gas-producing organisms. **Gallbladder perforation** is an uncommon but life-threatening complication of cholecystitis. Gangrenous

TABLE 79-4 Complications of Acute Pancreatitis

Pancreatic	Peripancreatic	Extrapancreatic	
Fluid collection	Fluid collection	*Cardiovascular*	*GI*
Necrosis	Necrosis	Hypotension	Peptic ulcer disease/erosive gastritis
Sterile or infected	Intra-abdominal or retroperitoneal hemorrhage	Hypovolemia	GI perforation
Acute or walled off	Pseudoaneurysm (of contiguous visceral arteries, e.g., the splenic)	Myocardial depression	GI bleeding
Abscess	Bowel inflammation, infarction, or necrosis	Myocardial infarction	Duodenal or stomach obstruction
Ascites	Biliary obstruction with jaundice	Pericardial effusion	Splenic infarction
	Splenic or portal vein thrombosis	*Pulmonary*	*Renal*
		Hypoxemia	Oliguria
		Atelectasis	Azotemia
		Pleural effusion (with or without fistula)	Acute renal failure
		Pulmonary infiltrates	Thrombosis of renal artery or vein
		Acute respiratory distress syndrome	*Metabolic*
		Respiratory failure	Hyperglycemia
		Hematologic	Hypocalcemia
		Disseminated intravascular coagulation	Hypertriglyceridemia