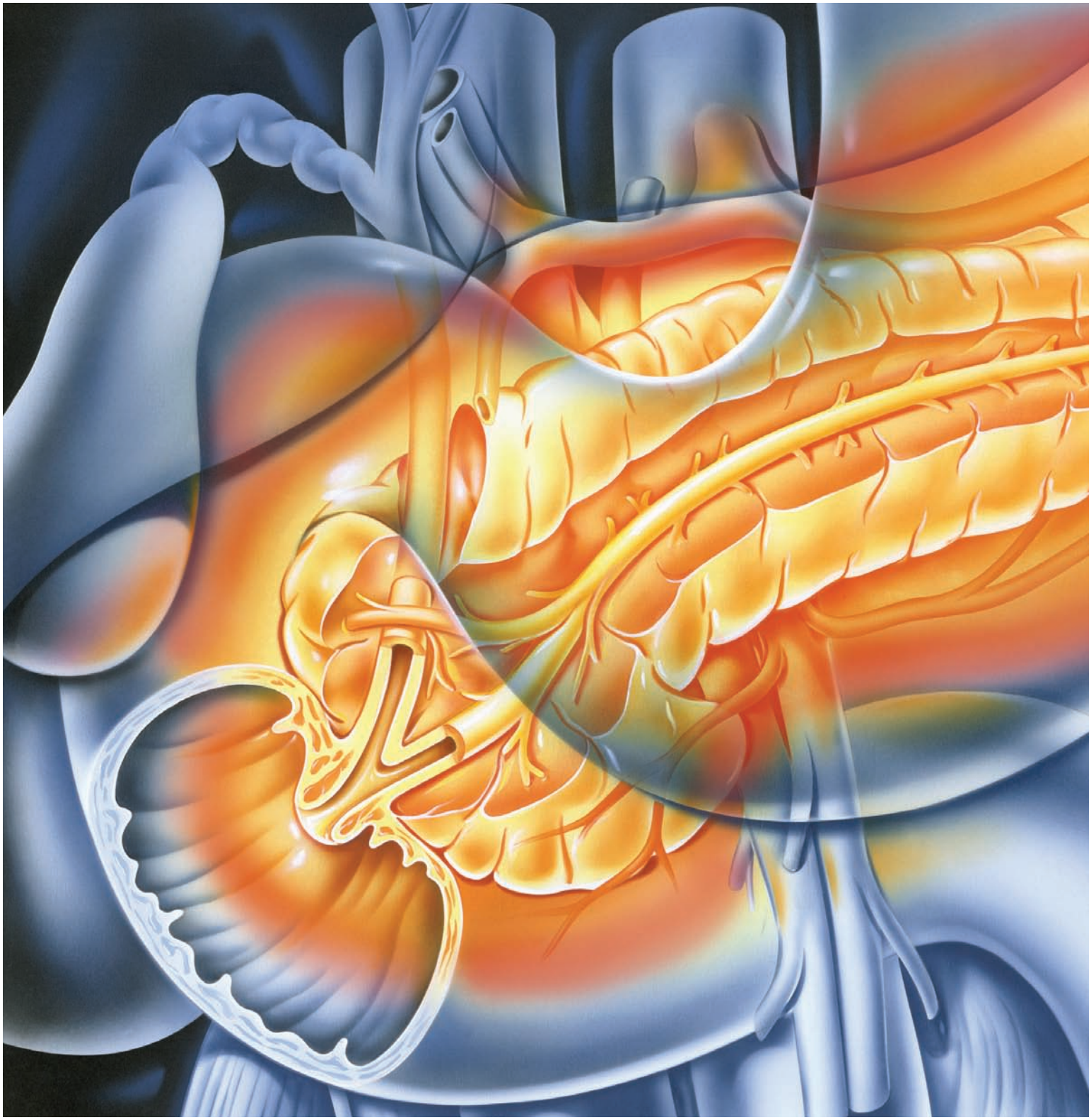

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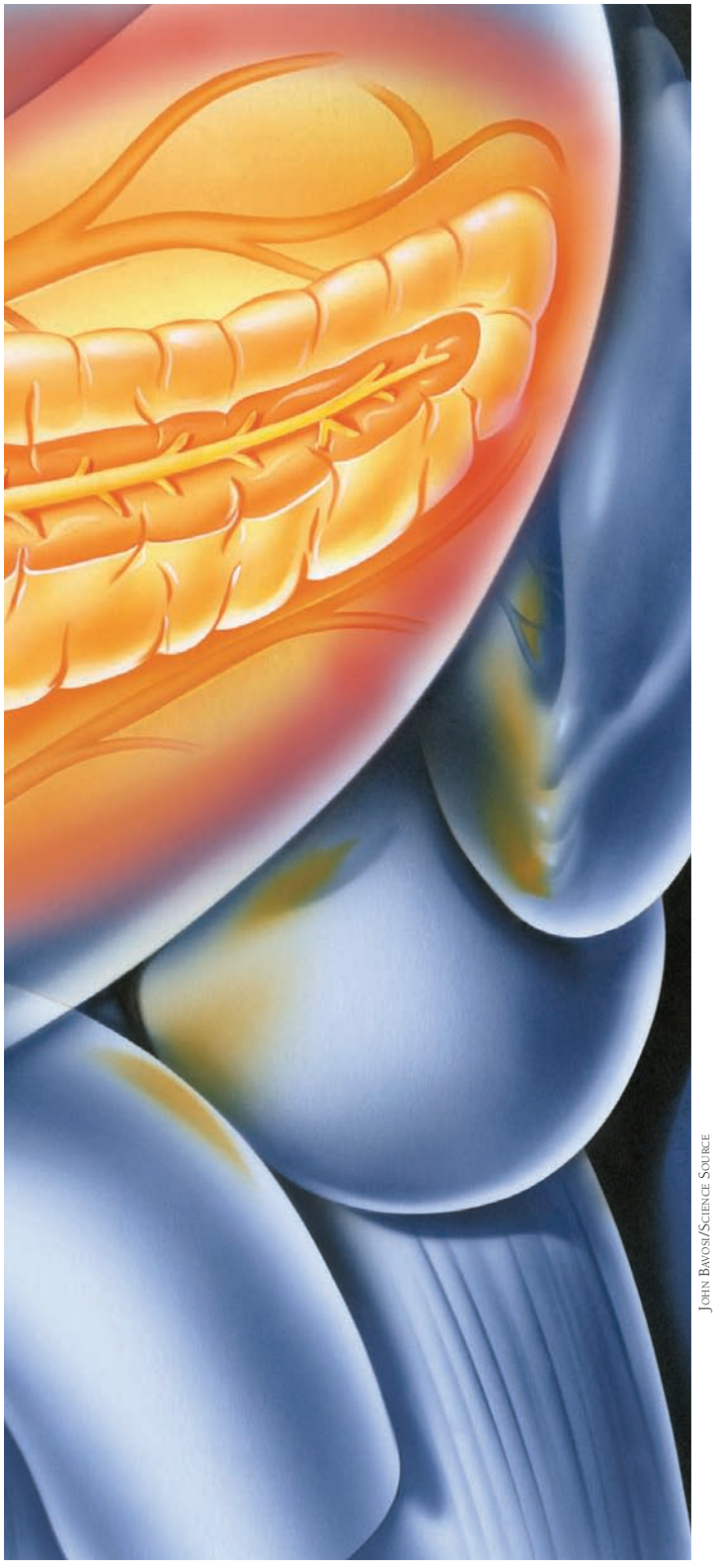
Understanding acute pancreatitis



JOHN BAVOSI/SCIENCE SOURCE

By Maureen E. Krenzer, MS, RN, ACNS-BC

MR. W, 56, ENTERS THE ED reporting severe abdominal pain that started suddenly after his evening meal. He describes it as constant and radiating to his back. He rates his pain as an 8 on a 0-to-10 pain intensity rating scale. He tells the ED nurse who takes his history that he's "probably been drinking too much" since he lost his job a year ago.

Lab tests reveal elevated levels of serum amylase and lipase. He's N.P.O. and receiving I.V. fluids at 150 mL/hour. An abdominal ultrasound is negative for gallstones, but a computed tomography (CT) scan of the abdomen shows pancreatic inflammation. The patient is admitted and the facility's protocol for acute alcohol withdrawal is put into effect.

Mr. W has classic signs and symptoms of acute pancreatitis. The onset is usually abrupt, often occurring after a large meal or excessive alcohol use. Patients typically have severe, constant pain in the midepigastric region that radiates to the back and flanks.

Although common, acute pancreatitis is potentially life-threatening, especially if complications occur. This article discusses the latest clinical information about pancreatitis and describes appropriate nursing interventions and patient teaching.

Inflammation triggers autodigestion

Besides producing insulin, the pancreas secretes digestive enzymes. Acute pancreatitis is characterized by pancreatic inflammation resulting in autodigestion, or the breakdown of pancreatic tissue by its own activated enzymes. (See *Pathogenesis of acute pancreatitis*.)

Normally, pancreatic enzymes don't become active until they leave the pancreas. During pancreatitis, however, inflammation delays release of activated enzymes, giving them time to attack pancreatic cells and leak into surrounding tissues. This process causes more inflammation.

The damage in acute pancreatitis occurs in stages:[1]

- Premature activation of trypsin, amylase, lipase, and other enzymes occurs inside the pancreas.
- Intrapancreatic inflammation through the release of cytokines and other proinflammatory mediators leads to micro- and macrovascular failure as the activated digestive enzymes autodigest the pancreas and peripancreatic tissues.
- In the last stage, extrapancreatic or systemic inflammation and injury may occur.

In mild pancreatitis, the stages are less severe and self-limiting.

Gallstones and chronic alcohol abuse account for 90% of acute pancreatitis cases.[2] When a gallstone lodges in the common bile duct, the obstruction raises pancreatic ductal pressure and leads to inflammation and rupture of the small pancreatic ducts, resulting in premature activation of pancreatic enzymes. Alcohol increases the tendency for production of pancreatic secretions and formation of protein plugs within the pancreatic ducts. The multifactorial effect of alcohol on the acinar cells may obstruct the flow of pancreatic secretions and lead to inflammation and pancreatic damage. Spasms of the sphincter of Oddi may also lead to inflammation, although these results have been controversial.[1]

Acute pancreatitis in pregnancy is a rare event but can lead to preterm labor and delivery.[3] (See *What can trigger acute pancreatitis?* for more potential causes).

Classifying types of pancreatitis

The most common type of pancreatitis is *mild acute pancreatitis*, also called interstitial or edematous pancreatitis. Areas of fat necrosis and interstitial edema develop in and around the pancreas (the peripancreatic tissues). Mild acute pancreatitis is usually self-limiting without organ failure or local or systemic complications.

Severe acute pancreatitis, also known as necrotizing pancreatitis, is a life-threatening disorder associated with local and systemic complications including intra-abdominal infections, pancreatic necrosis, and organ failure.[4] An additional type, *moderately severe acute pancreatitis*, is described as transient organ failure or local or systemic complications. (See *Severity of pancreatitis* for a breakdown of levels.)

Key assessment findings

The most common symptom of acute pancreatitis is a sudden onset of sharp, twisting, deep, upper abdominal pain. It frequently radiates to the back and is associated with nausea and vomiting. Localized pain commonly reflects mild acute pancreatitis, while more diffuse pain may be associated with severe or necrotizing pancreatitis. The degree of pain may or may not reflect the severity of the disease process.

Other common signs and symptoms of acute pancreatitis are anorexia, hypoactive bowel sounds, upper abdominal tenderness without rigidity, abdominal distension, and diarrhea. In more severe cases, the

Pathogenesis of acute pancreatitis

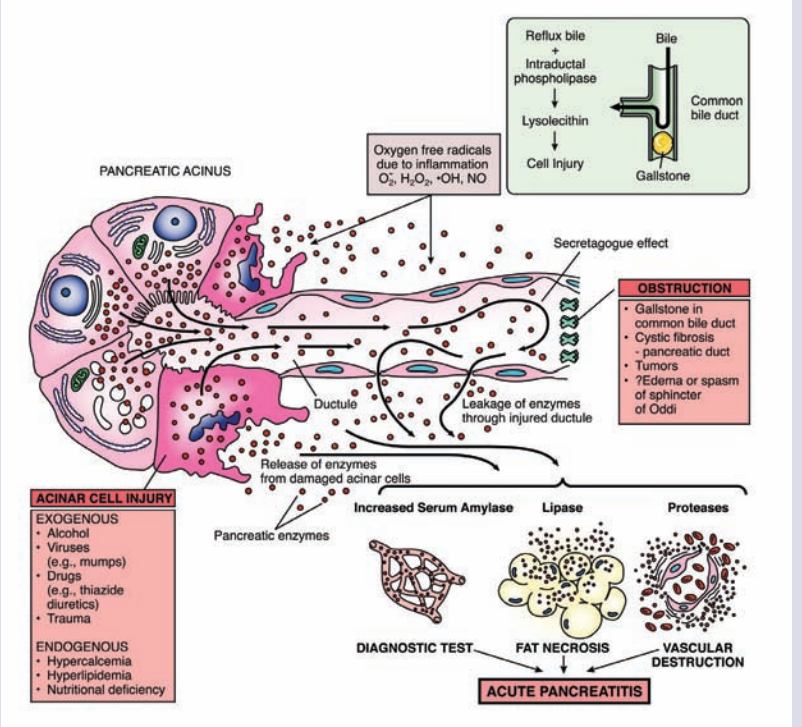


Image from Rubin E MD and Farber JL MD. *Pathology*, 3rd Edition. Philadelphia: Lippincott Williams & Wilkins, 1999.

patient may have fever, tachycardia, hypotension, weakness, diaphoresis, shock, jaundice, and peritoneal signs, such as guarding and rebound tenderness.[5,6] An abdominal mass may be palpable due to the enlarged pancreas.

A diagnosis of acute pancreatitis requires at least two of the following:[7]

- characteristic abdominal pain
- serum amylase and/or lipase level at least three times the upper limit of normal. These enzymes leak into the blood as pancreatic cells are damaged. (See *Serum lab testing for acute pancreatitis*.)
- characteristic findings from abdominal imaging (CT scan, magnetic resonance imaging, or ultrasound).

What diagnostic tests reveal

The healthcare provider may order these tests to examine the pancreas and surrounding region.

- An abdominal ultrasound can help determine the presence of gallstones quickly, noninvasively, and without radiation.
- An abdominal CT scan is usually performed with contrast to confirm an unclear diagnosis and to evaluate the extent of damage to the pancreas and surrounding region. The nurse should evaluate the patient's renal function and assess for a history of adverse reactions to contrast media.
- Endoscopic retrograde cholangiopancreatography (ERCP), an invasive diagnostic procedure, uses an endoscope to view the ampulla of Vater, pancreas, and bile ducts. ERCP requires contrast medium and fluoroscopy. An advantage of ERCP is that it can also be used to remove obstructions such as gallstones, perform biopsies, and/or insert a stent to promote drainage.
- Magnetic resonance cholangiopancreatography (MRCP) is a noninvasive procedure that allows visualization of the biliary tree and pancreatic ducts. This doesn't involve contrast media and is less risky for the patient than ERCP. MRCP may be an alternative for a patient with a history of adverse reactions to contrast media.

The treatment plan after diagnosis depends on whether pancreatitis is mild or severe. Making this determination can be challenging. Multiple scoring systems exist that may help to predict outcomes of acute pancreatitis but many of them, such as the Ranson criteria, require 48 hours for additional blood work and complete assessment. The Bedside Index of Severity in Acute Pancreatitis (BISAP) and the Harmless Acute Pancreatitis Score (HAPS) may be helpful during initial assessment for severity in the ED.[8] BISAP uses vital signs, mental status, age, blood urea nitrogen (BUN), white blood cell count, and pleural effusion detected on imaging; all usually completed on presentation to ED.[9] HAPS uses absence of rebound tenderness, creatinine < 2 mg/dL, and normal hematocrit to predict mild cases of acute pancreatitis.[8] Looking at factors such as age, body mass index, comorbidities, lab

What can trigger acute pancreatitis?[12,13,16,17]

- alcohol abuse
- gallstones, microlithiasis, or biliary sludge obstructing the common bile duct
- drugs such as acid-suppressing medications, immunomodulators, diuretics, antimicrobials, nonsteroidal anti-inflammatory medications
- hypercalcemia
- hypertriglyceridemia (>1,000 mg/dL)
- idiopathic; may also develop during pregnancy and postpartum period
- infections: viral: mumps, coxsackie virus, cytomegalovirus, HAV, HBV, HIV/AIDS; bacterial: mycoplasma, legionella, salmonella; fungal: aspergillus, candida albicans; parasitic: toxoplasma, cryptosporidium
- inflammatory bowel disease
- pancreas divisum
- pancreatic tumors
- peptic ulcer disease
- parenteral nutrition
- scorpion envenomation
- trauma: blunt or penetrating abdominal trauma; post-ERCP; surgical trauma
- toxins
- hypothermia.

Severity of pancreatitis[15]

Mild acute pancreatitis

- No organ failure
- Lack of local or systemic complications

Moderately severe acute pancreatitis

- Organ failure that resolves in 48 hours (transient)
- Local or systemic complications without persistent organ failure

Severe acute pancreatitis

- Persistent single or multiple organ failure (> 48 hours).

Serum lab testing for acute pancreatitis[14]

In addition to amylase and lipase, additional lab testing may include:

- complete blood cell count with differential
- electrolytes
- BUN, creatinine, glucose
- coagulation studies
- liver enzymes
- lactate
- albumin.

values, and systemic inflammatory response syndrome (SIRS) to determine those most at risk for developing organ failure is recommended.[4] (See *Understanding SIRS criteria.*)

After the patient has been diagnosed with acute pancreatitis, the nurse should frequently check vital signs and lab values. Likely findings include an increased white blood cell (WBC) count, tachycardia, and fever due to the inflammatory process. Mild confusion and hypoxemia are also common, the patient's pulse oximetry should be monitored and supplemental oxygen provided if indicated.

Managing the acute episode

Treating an episode of acute pancreatitis includes supportive care and curative interventions when possible. The first goal is to stabilize the patient hemodynamically. Administer I.V. crystalloids such as lactated Ringer's solution or colloids and plasma expanders for more critically ill patients, as prescribed. Early aggressive I.V. hydration has been associated with less complications and improved outcomes.[4] Monitor vital signs, oxygen saturation, pain intensity level, intake and output, hematocrit, and BUN levels to help assess hemodynamic stability. A patient with severe acute pancreatitis may need support with an inotropic drug such as I.V. dobutamine, hemodynamic monitoring, mechanical ventilation, and monitoring of intra-abdominal pressure via the bladder for development of abdominal compartment syndrome.

As prescribed, manage pain with an opioid such as fentanyl or hydromorphone. Besides making the patient more comfortable, these drugs decrease pancreatic enzyme secretion. Consistently assess the patient's pain using the same pain intensity rating scale each time to determine changing patterns of pain and evaluate the patient's response to treatment.

Reducing the stimulation of pancreatic enzyme release as much as possible reduces inflammation and autodigestion until the pancreas heals. Eating stimulates pancreatic secretions, so keep the patient N.P.O. until nausea and vomiting have resolved, serum amylase levels start returning to normal, and pain subsides. Although still controversial, the type of diet to resume can be anything from clear liquids to a soft low-fat diet and does not require a stepwise approach.[7] In mild acute pancreatitis, this may take 3 to 7 days. In severe pancreatitis, recovery may take up to 7 weeks. The patient may need a nasogastric tube to manage vomiting or paralytic ileus.

Severe acute pancreatitis is a hypermetabolic and hypercatabolic event that requires nutritional support. Enteral feeding via a nasogastric or jejunal tube is safe and recommended for those requiring nutritional support. Enteral feeding stabilizes gut bacteria, which prevents bacterial translocation and subsequent infection, and is more physiologic than parenteral nutrition (PN). PN should be used as a second line for those who can't meet nutritional requirements or tolerate enteral feeding.[7]

Treating acute pancreatitis also includes addressing the precipitating event. If the patient has mild gallstone pancreatitis, for example, cholecystectomy during this admission is recommended.[10] If the patient has an obstructing gallstone, the pancreatic inflammation may need more time to resolve with conservative treatment, so the patient may be discharged home with a plan for follow-up surgery when the acute inflammatory issues are resolved. If the patient continues to experience severe pain, the obstruction may be relieved via ERCP, but many patients have recurrent episodes and require subsequent gallbladder removal.[7] For patients with acute cholangitis, urgent ERCP and sphincterotomy may be necessary.[6]

Possible systemic complications

Patients with severe acute pancreatitis are at risk for various systemic complications. (See *On guard for complications of severe acute pancreatitis.*) For example, most patients with severe acute pancreatitis experience hypoxemia or other respiratory issues in the first 2 days.[11] Risk factors such as obesity, age (>55), medical comorbidities, presence of hypovolemia, SIRS, mental status changes on presentation, pleural effusion or infiltrates, and multiple or extensive fluid collections outside of the pancreas are associated with development of severe disease.[12]

Routine nursing care should include the following:

- Monitor patients for early signs and symptoms of complications, such as increased abdominal pain and tenderness, fever, and WBC count. Changes in mental status are early signs of impending respiratory failure or shock.
- Assess for a bluish discoloration around the umbilicus (Cullen sign) or a reddish-brown discoloration along the flanks (Grey-Turner sign); these may indicate hemorrhagic pancreatitis or expelled exudates from necrotic areas. Notify the healthcare provider immediately if you see these signs.

Understanding SIRS criteria[4]

SIRS is defined as two or more of the following:

- temperature, < 36° C (96.8° F) or > 38° C (100.4° F)
- respiratory rate > 20 breaths/minute or $Paco_2$ < 32 mm Hg
- pulse > 90 beats/minute
- WBC count < 4.0 or > 12.0 cells/mm^3 or > 10% immature neutrophils (bands).

• Assess for dizziness or lightheadedness and hypotension. Cardiac involvement can result from the release of myocardial depressant factor and hypovolemic shock, which decreases cardiac output.
• Document intake and output and assess for signs and symptoms of fluid overload, including dyspnea, peripheral edema, crackles in the lung fields especially the bases, and electrolyte imbalances. Gastrointestinal (GI) losses from vomiting and diarrhea, if present, can lead to significant electrolyte imbalances such as hypokalemia and hypomagnesemia.
• Document urine/stool characteristics; as the kidneys excrete more bile into the urine (when pancreatitis is caused by obstructive biliary stones), urine may become brownish and foamy. Stool often becomes foul-smelling and pale or gray due to the lack of bile entering the GI tract because of the biliary obstruction.
• Monitor intra-abdominal pressure for development of abdominal compartment syndrome.
• Watch for Chvostek or Trousseau signs as indicators of hypocalcemia. (See *Reading the signs of hypocalcemia*.)

On guard for complications of severe acute pancreatitis[13]

- Respiratory failure can occur secondary to hemodynamic instability and as a result of pancreatic phospholipase A, which destroys the lipid component of surfactant.
- SIRS develops from the premature activation of pancreatic enzymes, humoral factors such as myocardial depressant factor, platelet-activating factor and other proinflammatory cytokines, and neutrophils.
- Hypovolemic shock occurs due to the intravascular fluid shifts from the combination of vasodilation and increased vascular permeability. Hypovolemic shock can also occur as a result of hemorrhage.
- Acute tubular necrosis is fairly common, resulting from renal ischemia secondary to hypotension and hypoxemia.
- Decreased level of consciousness is related to pancreatic encephalopathy.
- Disseminated intravascular coagulation occurs.
- Metabolic changes such as hyperglycemia, metabolic acidosis, and hypocalcemia can occur.
- Intra-abdominal hypertension and abdominal compartment syndrome can occur due to the combined effect of the inflammatory process and aggressive fluid resuscitation. Increased intra-abdominal pressure can shift the hemi-diaphragm up, leading to further atelectasis.

Detecting localized complications

Localized complications of pancreatitis include peripancreatic fluid collection, acute necrotic collection, walled-off necrosis, and pseudocyst.
• Peripancreatic fluid collections are associated with interstitial edematous pancreatitis without necrosis and occur typically in the first 4 weeks. Fluid collections are often left untreated to resolve on their own.
• Acute necrotic collection occurs from severe autodigestion in necrotizing pancreatitis and includes some fluid and necrosis in the pancreas or peripancreatic tissues.
• Walled-off necrosis is a mature encapsulated collection of necrosis either within the pancreas or in the surrounding tissues. This can occur 4 weeks or more after initial presentation of necrotizing pancreatitis. Necrosis can be sterile or infected.
• Pseudocyst is a collection of pancreatic enzymes and possibly minimal necrotic tissue and/or blood enclosed by surrounding tissues. This typically occurs outside the pancreas more than 4 weeks after initial presentation of interstitial pancreatitis.

Routine prophylactic antibiotics for patients with severe acute pancreatitis isn't recommended.[4] If indicated for infected necrosis, antibiotics should be specific to the bacteria identified. Using CT-guided percutaneous aspiration, the healthcare provider can obtain fluid samples for cultures or analysis when needed to identify suspected infected necrosis or to evaluate a patient who fails to improve. Percutaneous or endoscopic drainage of infected walled-off necrosis is preferred in stable patients versus surgical intervention.

Patient education

When gallstone pancreatitis is being managed conservatively, provide written dietary instructions and teach patients to follow a low-fat diet to reduce gallbladder stimulation and to notify their healthcare provider at the first sign of recurrent pain, nausea, or vomiting. Refer patients who have a history of significant alcohol intake to case management or social work, so they can provide counseling about alcohol cessation programs and offer to help the patient get started with

Reading the signs of hypocalcemia

In pancreatitis, the pancreas' necrotic enzymatic action uses up the calcium, leading to hypocalcemia. These signs may indicate hypocalcemia.

Chvostek sign: Tap the patient's facial nerve directly in front of the ear. A positive sign is present when the facial muscles contract on the same side of the face as the tapping.

Trousseau sign: Inflate a BP cuff on the upper arm to a level above the patient's systolic BP for 2 minutes. A positive sign is present when the hand spasms and flexes in response to the test.

enrollment efforts. Screening the family for needs related to alcoholism may also be indicated.

Road to recovery

Mr. W's acute mild pancreatitis resolves without complications. He resumes eating on day 4 and is discharged on day 6. He and his wife are referred to community programs for alcohol addiction, job retraining, and stress management. Prompt assessment and interventions helped Mr. W recover fully and avoid life-threatening complications of acute pancreatitis. ■

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