The Patient with Pancreatitis

CE110-60 :: 1.00 Hours

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Objectives
The goal of this program is to update nurses' ability to care for patients with pancreatitis. After you study the information presented here, you will be able to —

- List two causes and two complications of acute and chronic pancreatitis.
- Describe the typical presentation of a patient with acute pancreatitis.
- Differentiate between the pain management of patients with acute and chronic pancreatitis.

A very anxious, 50-year-old Maria was crying and doubled over in pain as she entered the ED. She complained of a progressively worsening epigastric distress and a lack of appetite for two days. When her nurse, Alice, asked her when she had last eaten, Maria stated that she hadn’t had any food for “a few days” and that “even beer” made her nauseous. Upon physical assessment, Alice observed a classic combination of signs and symptoms: clammy skin; a mildly distended abdomen; guarding; a nonradiating, gnawing pain in the midepigastric area and midback; and hypoactive bowel sounds. Although she suspected pancreatitis, Alice noted that ecchymosis on the flank area (Grey Turner’s sign) and umbilical area (Cullen’s sign), which often indicate hemorrhagic pancreatitis, were absent. Knowing that such pain may impair respiratory function, the nurse carefully listened for any abnormal breath sounds. Maria’s breathing was tachypneic and shallow, but her breath sounds were clear. Her blood pressure was 150/90 mm/Hg, heart rate 105 beats/min, respirations 24/min, and temperature 99.9 F.

Pancreatitis can affect anyone at any age. It can cause critical illness and, in the acute stages, may even be fatal. Nurses in every practice setting — from the ED to medical/surgical and critical care units to patients’ homes — need to be familiar with the identification and management of this disease. Nurses need to be able to identify the classic signs and symptoms of chronic and acute pancreatitis, underlying pathophysiology, usual treatment, and the steps to follow to achieve adequate pain control.

The pancreas, positioned in the abdominal cavity behind the stomach and between the spleen and duodenum, is both an endocrine and exocrine gland. Alpha, beta, and delta cells, located
in the islets of Langerhans on the surface of the pancreas, perform the endocrine function of
this gland by secreting hormones directly into the bloodstream. These hormones include:
glucagon (from alpha cells), which elevates serum glucose; insulin (from beta cells), which
controls metabolism of carbohydrates, proteins, and fats; and somatostatin (from delta cells),
which inhibits both glucagon and insulin secretions.¹

The beta cells also produce amylin, a pancreatic beta cell-secreted hormone that regulates
glucose balance in conjunction with insulin and glucagon in response to food intake. Amylin is
secreted with insulin and contributes more significantly to glucose lowering after meals (the
postprandial period), especially during the three hours immediately after eating. Amylin
slows the hepatic production of glucose and also serves as a satiety signal, sending the
message of fullness across the blood-brain barrier to encourage us to stop eating.²

In people with pancreatitis, amylin, like insulin, may not be adequately produced. Amylin is
especially effective in lowering postprandial blood glucose levels and tends to promote weight
loss.²

The pancreas connects to the digestive system at the level of the duodenum via the ampulla
of Vater, where the common bile duct meets the main pancreatic duct. This organ plays a
major exocrine role in the digestion of food by secreting the enzymes necessary to break
down carbohydrates, fats, and proteins. These enzymes combine with sodium bicarbonate to
form pancreatic juice, which is secreted when chyme (partially digested food) is present in
the duodenum. (For a diagram of the pancreas, go to www.cancer.gov/cancertopics
/pdq/treatment/pancreatic/Patient).

**Acute Pancreatitis:** The activation of pancreatic enzymes within the pancreas leads to
autodigestion of the pancreas and any other organ with which the enzymes come in contact.
The most common cause of acute pancreatitis is gallstones, which occur more frequently in
women, especially between the ages of 50 and 60 years.¹³ Alcohol is the second most
common cause. This occurs more often in men who are in their early 30s. These two
conditions account for 60% to 80% of the cases of acute pancreatitis. Other causes include
⁻¹,⁻³,⁻⁴

- Postoperative complications (surgical or endoscopic, especially after endoscopic
  retrograde cholangiopancreatography [ERCP])
- Peritoneal dialysis
- Parasitic, viral, and bacterial infections
- Toxins
- Pregnancy
- Carcinoma
- IV lipid infusions
- Recent blunt trauma
- Hypertriglyceridemia
- Peptic ulcer disease
- Cystic fibrosis

An increased risk for developing pancreatitis has also been found among individuals who
have hereditary angioedema. These individuals are likely to present with all the symptoms
seen with a typical episode of angioedema (swollen face and tongue), but they will also
experience severe abdominal pain because their pancreatic ductal drainage system will also
be swollen and blocked.⁵ While diabetes mellitus often develops as a result of pancreatitis,
evidence is emerging that points to Type 2 diabetes as increasing the risk for development of
acute pancreatitis. One study of over 337,000 patients with diabetes demonstrated a
2.83-fold greater risk for acute pancreatitis when compared to over 337,000 patients without
diabetes.⁶ (LEVEL B)

Emerging evidence from research also points to autoimmune processes playing a much more
important role than previously thought. Pancreatitis can also be associated with multiple autoimmune disorders, such as systemic lupus erythematosus (SLE) and rheumatoid arthritis. When patients with underlying autoimmune disorders have abdominal surgical procedures performed, they are at a greater risk for developing pancreatitis postoperatively.

Patients with autoimmune pancreatitis tend to have more extrapancreatic complications than patients with pancreatitis triggered by nonautoimmune causes, including biliary tract involvement and pulmonary complications. Patients with autoimmune pancreatitis are also more likely to be misdiagnosed with pancreatic cancer.

Pancreatitis is also associated with drugs such as glucocorticoids, thiazide diuretics, acetaminophen, estrogen, chemotherapeutic agents, and HIV treatments with a combination of didanosine (Videx) and tenofovir (Viread). Recently, atypical antipsychotics, such as olanzapine (Zyprexa), risperidone (Risperdal), and clozapine (Clozaril), have been associated with the development of pancreatitis. Valproic acid (Depakene) use also increases the risk for pancreatitis, especially among children. There have also been reports of patients developing severe pancreatitis following vaccination with the combined hepatitis A and B vaccines.

Patients with acute disease complain of constant, gnawing, epigastric pain that frequently radiates to the back. They often have nausea, vomiting, and a low-grade fever. Initially, pancreatic enzymes are released, leading to tissue injury and destruction.

The inflammatory response is activated and chemical mediators are released, which trigger events, such as vascular permeability, which causes vascular volume loss, hypoperfusion, and bleeding within the pancreas. The pancreas may become necrotic secondary to hypoperfusion, known as necrotizing pancreatitis or severe acute pancreatitis. The necrotic area can become infected, releasing endotoxins into the bloodstream and causing sepsis, shock, adynamic ileus, disseminated intravascular coagulation (DIC), adult respiratory distress syndrome (ARDS), and acute renal failure (ARF), all of which are imposed upon the underlying electrolyte imbalance and acid-base abnormalities that develop with acute pancreatitis. These life-threatening complications occur in about 20% to 30% of patients. Hemorrhagic pancreatitis and an intra-abdominal bleed may also ensue.

Fear of death is common and understandable in the patient with acute pancreatitis. Acute pancreatitis can lead to the development of chronic pancreatitis from repeated scarring and obstructions. Patients with severe acute pancreatitis are at high risk to develop organ failure, which is their major cause of death. Evidence is emerging that organ failure can be predicted by monitoring the levels of interleukin-10 (IL-10) and IL-6 — inflammatory cytokines (proteins that transmit genetic symbols), which, when released into the system after stress, provoke a “flight or fight” response in the adrenal cortex. Those with poor health and multiple disorders before developing pancreatitis tend to have a more protracted and poorer course, especially when surgical intervention is required. Children, however, fare much better, even if they require surgical intervention.

**Chronic Pancreatitis:** Chronic pancreatitis may be a persistent condition or a series of acute episodes that are superimposed on a previously damaged organ. The causes of chronic pancreatitis are similar to acute, with the major addition of cystic fibrosis in children and malnutrition in patients outside the United States. Among the usual causes of chronic pancreatitis — acute pancreatitis, genetic malformations, obstructed ducts, and chronic heavy alcohol use — alcoholism is the most common. As with some other chronic diseases, there are idiopathic, hereditary, congenital, and autoimmune forms.

Chronic pancreatitis produces necrotic-inflammatory changes that result in irreversible damage of the gland, malnourishment, and diabetes mellitus, but the common denominator
is destruction of a much-needed organ through autodigestion and scarring.¹

Those with chronic pancreatitis experience weight loss, malnourishment, and steatorrhea (fatty stools) due to malabsorption. In later stages, pain diminishes with the death of pancreatic tissue and diabetes mellitus will ensue. Other complications may include —

- Sepsis
- Shock
- Pseudocyst
- Abscess or fistula formation
- Biliary stenosis

Hypocalcemia, GI bleeding, renal failure, and encephalopathy may also develop.²¹ Treatment for chronic and acute pancreatitis varies according to the cause and presenting symptoms.

**Nailing the diagnosis**

Serum amylase, lipase, and trypsin are lab tests specifically used to aid diagnosis of pancreatitis.¹ A trypsin assay is probably the most accurate serum indicator for acute pancreatitis. However, it is not readily available, so it isn’t used routinely.

Elevated serum amylase and lipase levels are common indicators of pancreatitis. However, the elevation does not indicate the severity of the disease, and mild to moderate abnormal values may be attributed to other glands. The serum amylase level starts to rise two hours to 12 hours after the onset of symptoms, peaks within 12 hours to 72 hours of the onset of symptoms, and returns to normal within one week.²¹ Although its specificity is only 20% to 60%, serum amylase is the most widely used method for diagnosing pancreatitis. In some cases of alcohol-induced chronic pancreatitis, amylase levels may not rise at all. Serum lipase levels increase within four hours to eight hours, peak at about 24 hours, and remain elevated for up to eight days to 14 days.²¹ The specificity of serum lipase level is 99%, making it a more accurate indicator of acute pancreatitis than serum amylase. Another useful marker for identifying both acute and chronic pancreatitis is glycoprotein 2 (GP2), a diagnostic test, which is available but not widely used.²⁰

Two new diagnostic tests, while not yet widely used or available, can assist in pancreatitis diagnosis and monitoring of pancreatic function. A point-of-care urine test that assesses the level of trypsinogen in only three minutes has proven to be an effective rapid assessment tool in the ED setting.²² A fecal test for pancreatic elastase has also been shown effective for long-term follow-up assessments for patients with severe pancreatic insufficiency.²³

A biological connection has been established between the effect of stress and the development of pancreatitis. Patients with acute pancreatitis have been found to have extremely high levels of serum interleukin-18 (IL-18) — an inflammatory cytokine (a protein that transmits genetic symbols), which, when released into the system after stress, provokes a “flight or fight” response in the adrenal cortex.²⁴ The levels of IL-18 correlate directly with the severity of pancreatitis — those with lower levels of IL-18 had less severe pancreatitis, while those with higher levels had more severe pancreatitis.²⁴

Other findings may include elevated white blood cell counts, glucose, and hematocrit levels, and low serum levels of albumin, potassium, calcium, and magnesium.¹ Because other conditions, including renal failure, alter amylase and lipase values, other diagnostic studies are indicated.

An abdominal X-ray can rule out conditions that mimic acute pancreatitis, such as a small bowel obstruction, or confirm chronic pancreatitis, when pancreatic calcifications are seen. A gas-filled sentinel loop of the proximal small bowel in close proximity to the inflamed
pancreas is a specific finding for pancreatitis. An ultrasound can help to visualize the sludge, dilated ducts, and gallstones of biliary-induced pancreatitis, but can be limited by intestinal gas and adipose tissue. CT can expose a fatty liver, which often accompanies alcoholic pancreatitis, inflammatory changes, pancreatic masses, necrosis, and/or fluid collections. An ERCP allows visualization of the duodenum, common bile duct, and other pancreatic structures, and diagnosis of pancreas divisum or sphincter of Oddi disease, but poses its own risk of ERCP-induced pancreatitis from blunt trauma during instrumentation. Endoscopic ultrasonography (EUS) is a useful test for small stones, tumors, or biliary sludge.

Maria’s ultrasound is negative for gallstones and she is scheduled for a CT scan of the abdomen with contrast dye. This test reveals a slightly enlarged liver with many fatty deposits and pancreatic calcifications. These results, added to her history, physical assessment, and lab findings, are consistent with alcohol-induced pancreatitis. She is sent to a med/surg floor for supportive treatment.

Supportive Care

An episode of pancreatitis is managed by resting the pancreas, treating the underlying problem, and administering analgesics until recovery takes place. IV fluids, daily weights, and accurate intake and output monitoring are important components of initial treatment. Patients are given nothing by mouth to avoid stimulation of the pancreatic enzymes. A nasogastric tube (NGT) is not usually inserted unless there is vomiting, gross abdominal distention, an ileus, or an obstruction. Enteral feeding via a small-bore feeding tube inserted into the jejunum below the ligament of Treitz is being used in lieu of or in conjunction with parenteral nutrition. Total enteral nutrition maintains the gut integrity, and there is evidence that enteral feeding reduces complications and improves outcomes for patients with severe pancreatitis. If pain or vomiting develops while enteral nutrition is infusing, the volume should be decreased and total parenteral nutrition (TPN) used as a supplement. Antiemetics can also be used.

Total parenteral nutrition has shown no benefit in the treatment of pancreatitis, unless the patient is without enteral feedings for seven to 10 days. In that case, triglyceride levels should be monitored and maintained below 500 mg/dL to reduce the risk of total parenteral nutrition -induced pancreatitis. When total parenteral nutrition is used, research indicates that supplementation with glutamine can enhance immune function and the healing process.

When eating resumes, a low-fat, low-protein diet with oral enzymes, such as pancreatin (Creon) or pancreatic lipase (Viokase or Pancrease), and a daily multivitamin regimen are prescribed. Supplementation with antioxidants, such as vitamins C and E, has also been beneficial. Evidenced-based guidelines for nutritional aspects of caring for patients with pancreatitis can be found at www.espen.org/espenguidelines.html.

Monitoring for Complications

Close observation is critical for the recovery of these patients who are at risk for developing life-threatening complications, including septic and hypovolemic shock. Nurses need to monitor daily lab values for signs of infection, renal failure, and hemoconcentration — rising hemoglobin and hematocrit that might represent third spacing, which can lead to pancreatic hypoperfusion, hypovolemia, and shock. Frequent lung assessments and encouraging coughing and deep breathing are essential to combat development of pleural effusion and pneumonia. Monitor the patient’s flank area and periumbilical areas for ecchymosis that accompanies hemorrhagic pancreatitis. Measure abdominal girth daily for indication of distention and ascites, and assess bowel sounds for the presence of paralytic ileus. The patient should move about and perform range of motion exercises every two hours to
prevent skin breakdown from poor nutritional status and immobility due to pain. Check for pallor, jaundice, and ecchymosis that may indicate liver involvement, which may occur from obstructed bile ducts with biliary pancreatitis. Routinely assess for pain, which may be excruciating with acute pancreatitis. Moderate to severe pain is managed with opioid analgesics. Morphine is the drug of choice now, although it was previously thought to cause spasms of the biliary and pancreatic sphincters. Meperidine (Demerol) use has decreased dramatically because of the undesirable toxic metabolites that form. Positioning, such as flexing the knees toward the chest, diminishes pain by reducing the stretch and tension of the abdominal wall. Monitor for changes in mental status that may indicate alcohol withdrawal in patients with a history of alcohol abuse.

Deterioration frequently warrants transfer to critical care areas, where aggressive fluid resuscitation and hemodynamic pressure monitoring are necessary. Patients may need intense pulmonary care, including continuous oxygen saturation monitoring, chest physical therapy, bronchodilators, suctioning, and mechanical ventilation. Some may require continuous renal replacement therapy (CRRT) or dialysis, if ARF develops as a complication of hypovolemic (hemorrhagic pancreatitis) or septic (necrotizing pancreatitis) shock.

**Spontaneous Cure and Surgical Intervention**

Depending on the etiology, most cases of pancreatitis are treated with nothing more than supportive care; others need aggressive surgical intervention. To view evidence-based guidelines for the treatment of pancreatitis, go to: www.guideline.gov/summary/summary.aspx?doc_id=5512.

Interstitial or edematous pancreatitis, which involves a leakage of fluid into the interstitial space of the pancreas without interfering with the microcirculation and oxygenation, has a good prognosis with supportive treatment. Likewise, stones that block the ducts to the duodenum and cause a backflow of bile to the pancreas in acute biliary pancreatitis may pass spontaneously with only supportive treatment. Stones that do not pass must be removed endoscopically, surgically (open or laparoscope), or through lithotripsy. When pancreatic ducts are obstructed by calculi that do not resolve on their own, or are stenosed by scar tissue in chronic conditions, a stent is sometimes inserted via endoscopy to allow pancreatic secretions to drain into the duodenum. This procedure carries complications of its own, such as migration of the stent, abscess formation, stent-induced ulceration, pseudocyst, and ductal enlargement. A pseudocyst, a well-defined collection of sterile fluid that occurs two weeks or three weeks after an acute episode, may also resolve spontaneously or enlarge and cause a great deal of pain, necessitating surgical drainage. Pseudocysts can occur in and around the pancreas, as well as in the mediastinal area. An infected pseudocyst, or pancreatic abscess, also needs drainage, as well as antibiotic therapy.

On the other hand, necrotizing pancreatitis impairs circulation and results in patches of tissue death. Vasospasm also plays a role in the development of pancreatic ischemia and necrosis, especially in the early phase of acute necrotizing pancreatitis. Cases of sterile necrotizing pancreatitis, where there is no bacterial contamination, can be treated with supportive care. Surgical drainage may be considered in cases of persistent and severe pancreatitis. Infected or fulminating necrosis is one of the most serious complications of pancreatitis and carries a very high mortality rate. Clinicians use IV antibiotics that penetrate pancreatic tissue, such as imipenem (Primaxin), cefuroxime sodium (Zinacef), meropenem (Merrem IV), and ceftazidime (Ceptaz). Newer drugs effective for pancreatitis include linezolid (Zyvox), piperacillin/tazobactam (Zosyn), and the combination of IV mezlocillin (Mezlin) and metronidazole (Flagyl). Surgical debridement is also performed to combat bacteria that migrate from the intestine to the pancreas and peritoneal cavity. The timing for surgical intervention is not clearly established. One recent study compared mortality rates of those who had surgery within two weeks of admission and those who had...
surgery more than two weeks after admission. Percutaneous procedures performed early as well as laparoscopic debridement have also been found to result in more favorable outcomes, and mediastinal pseudocysts can also be treated effectively by endoscopy.27,30,31

The patient may return from surgery with an open wound with packing, which will need periodic debriding and clean packing; a closed wound and Penrose drains; or with a closed wound and continuous peritoneal lavage.3 Chronic disease can result in fistula formation between the pancreas and any surrounding organ and require surgery. Depending on the underlying problem, surgeries used to correct sequelae of chronic pancreatitis include a Whipple procedure (pancreatoduodenal resection), pancreaticojejunostomy, and pancreatic resection. These procedures often result in a complicated recovery, frequent hospital readmission, and a diminished quality of life.32

Dealing With Chronicity

The management of patients with chronic pancreatitis may last for years. Pain management for mild to moderate pain is initially treated with nonopioid analgesics, such as non-steroidal anti-inflammatory drug (NSAIDs), acetaminophen, and tramadol (Ultram), along with tricyclic antidepressants and selective serotonin reuptake inhibitor antidepressants (SSRIs). Opioid analgesics, such as morphine, hydromorphone (Dilaudid), and fentanyl (Duragesic), are used to control moderate to severe pain. Long-term pain management may necessitate surgical intervention. A bilateral splanchnicectomy may be performed to disrupt the neurological sensation pathway for pain perception from the pancreas. The procedure has been proven effective in controlling pain, while diminishing the need for opioids and improving the quality of life for people living with chronic pancreatitis.33

While the patient is hospitalized, some clinicians use cimetidine (Tagamet) to control the secretion of hydrochloric acid in the stomach and octreotide acetate (Sandostatin) to inhibit the release of pancreatic enzymes by manipulating the natural negative feedback system. By reducing pancreatic secretions, autodigestion of the pancreas is slowed and intrapancreatic pressure is lowered, which in turn reduces pain. Because a change in pain may herald a new complication, the etiology should be investigated before treatment. After discharge, pain is controlled through abstinence from alcohol; diet regulation; anti-inflammatory and analgesic drugs, such as oxycodone with acetaminophen (Percocet); and enzymes. Oral enzymes help to digest food, allow the pancreas to rest, and diminish pain.1 Nonenteric-coated enzymes that become active in the duodenum are thought to provide negative feedback that stops stimulation of pancreatic secretions, thereby decreasing pain. Sodium bicarbonate is given simultaneously to prevent their activation in the stomach from hydrochloric acid. One regimen prescribes eight nonenteric-coated enzymes with each meal and at bedtime, accompanied by 650 mg of sodium bicarbonate at each meal and 1,300 mg at bedtime or an H-2 blocker, such as cimetidine (Tagamet), ranitidine (Zantac), or famotidine (Pepcid), before and after each meal. Enzyme doses and schedules are adjusted to achieve pain control.

Prophylactic antibiotics are not generally given for either acute or chronic pancreatitis because they can enhance the colonization of resistant bacteria. Antibiotics are administered for secondary bacterial infections, pancreatitis caused by a bacterial infection, or necrotizing pancreatitis. Stool softeners are sometimes prescribed after an obstruction is ruled out to prevent constipation secondary to immobility and narcotic use.

Patients with chronic pancreatitis not only suffer from persistent pain and food intolerance, but they must cope with a lifestyle that often results in the loss of workdays and a disrupted family life. These stressors can worsen noncompliance in patients who may already be physiologically dependent on narcotics that do not completely control their pain. Patients with severe acute pancreatitis, as well as those with chronic pancreatitis, experience a diminished
Because alcohol is the most common cause of chronic pancreatitis in the United States, diagnosed patients need to be assessed for alcoholism. Support systems should be provided for families as well as the patients. In addition to the local support groups for those who abuse alcohol, free information for patients with chronic pancreatitis can be obtained by contacting the American Chronic Pain Association (ACPA), PO Box 850, Rocklin, CA 95677, (800) 533-3231, www.theacpa.org.

The treatment, teaching, and nursing care that is initiated in the hospital is often continued at home, and a visiting nurse may evaluate both the patient and family 24 hours to 48 hours after discharge. The nurse assesses the patient for complications, additional teaching needs, diet tolerance, psychosocial support, and pain control. Although complete absence of pain may not be an attainable goal, the nurse and patient can devise a plan that allows the patient to accomplish activities of daily living. If the patient is to be treated with home care nutritional support and/or IV antibiotics, the home health nurse can manage this care and initiate related teaching.

Three weeks after admission, Maria was sent home and visited by a home health nurse. The nurse found that Maria was afebrile and stable with no signs of infection, bowel obstruction, or pancreatic insufficiency. She reinforced hospital teaching about a low-fat diet and advised Maria to notify her physician if she experienced nausea, vomiting, or persistent diarrhea; a change in the amount, character, or location of pain; fever; or difficulty in breathing. The visiting nurse also reminded Maria that alcohol should be avoided and left the phone numbers of local support groups that could help her deal with alcohol abuse. The nurse evaluated whether the prescribed Percocet was adequately controlling Maria’s pain.

Maria was fortunate in that her support system, her husband and sister, showed concern for her well-being, and seemed knowledgeable and willing to help with her care. If she refrains from alcohol, Maria may recover completely and never experience another case of pancreatitis, or this may have been the first of many recurrent episodes of chronic pancreatitis.

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References


