ABSTRACT
This column discusses the pathophysiology, signs and symptoms, complications, treatments, and care of acute pancreatitis.

_Acute Pancreatitis is a rapidly occurring inflammatory process in which the pancreas simply turns on itself. There are two types of acute pancreatitis, interstitial and necrotizing. Interstitial pancreatitis is a mild inflammatory response and may be successfully managed in the hospital with bowel rest, hydration, pain control, and nutrition. On average, this type of acute pancreatitis lasts 3 to 5 days and is associated with minimal organ dysfunction, no necrotic injury, and an uneventful recovery process. About 80% of cases are interstitial, with a 5% to 10% mortality rate (Holcomb, 2007). The more severe form is known as necrotizing pancreatitis and because of complications, requires intensive care support and a long hospitalization. Necrotizing pancreatitis is differentiated by the life-threatening conditions of multisystem disease, necrosis, infection, and sepsis. Necrotizing pancreatitis occurs in about 20% of cases and has a substantially higher mortality rate of 50% (Holcomb, 2007)._

PATHOPHYSIOLOGY
Acute pancreatitis is caused by activation of blocked enzymes within the pancreas initiating the autodigestion process. In a normal situation, pancreatic enzymes are released from the pancreas in an inactive state and are activated in the duodenum. This premature activation of pancreatic enzymes results in inflammation and possible necrosis of the pancreatic endocrine and exocrine cells (Wrobleski, Barth, & Oyen, 1999).

Acute pancreatitis may lead to systemic complications because of the many vital functions performed by the endocrine and exocrine systems. The pancreas is a source for hormones that are important for regulating fuel metabolism. The key endocrine function of the pancreas is to secrete the hormones insulin and glucagon from the islets of Langerhans, which play a vital role in carbohydrate and lipid metabolism. The key exocrine function is to produce pancreatic juices that contain water, bicarbonate, and various digestive enzymes.

The leading causes of acute pancreatitis in adults are alcohol abuse and gallstones. Alcohol is a powerful stimulator of pancreatic secretions and directly irritates the pancreas, causing protein precipitates to form obstructing the acinar ductules and trapping the enzymes within the pancreas. Gallstones cause pancreatic duct obstruction as the stone migrates through the sphincter of Oddi. Bile is then refluxed into the pancreatic ducts, activating the enzymes within the gland. Both causes can lead to an acute pancreatic episode by blocking the enzymes within the pancreas initiating the autodigestion process (Lewis, Heitkemper, & Dirksen, 2004). Other causes of acute pancreatitis include endoscopic retrograde cholangiopancreatography, abdominal trauma, malignancy, hyperlipidemia, certain medications, and unknown factors (Burruss & Holz, 2005).

Autodigestion begins with the activated enzymes in the pancreas breaking down tissues and cell membranes. This process causes edema, hemorrhage, vascular damage, and fat necrosis within the pancreas (Burruss & Holz, 2005). The damage to the pancreas and surrounding tissues triggers the release of inflammatory mediators or cytokines. The escape of the activated enzymes and cytokines into the surrounding tissues and the abdominal cavity can lead to peritonitis. The enzymes and cytokines may then be absorbed from the abdominal cavity into the lymph vessels and eventually into the bloodstream. This process can result in damage to organs outside the abdominal cavity, leading to multisystem disease. Severe acute pancreatitis leads to an inflammatory response, vasodilation, hypotension, tachycardia, and bronchoconstriction, which are all signs and symptoms of septic shock (Holcomb, 2007).

SIGNS AND SYMPTOMS
Hallmark symptoms of acute pancreatitis are epigastric pain radiating to the back with nausea and vomiting (Cole, 2002). In necrotizing pancreatitis, there may be signs of retroperitoneal hemorrhage noted by Gray Turner’s and Cullen’s sign, respectively hemorrhagic discoloration of the flanks and umbilicus. Two key diagnostic tests involve serum amylase and lipase. These, along with other classic signs and symptoms of acute pancreatitis, can confirm disease (Burruss & Holz, 2005). These two enzymes produced by the pancreas usually increase on the first day of illness. Serum amylase decreases within 24 hours, whereas serum lipase, which is more specific for acute pancreatitis, remains elevated for at least 14 days (Holcomb, 2007). In cases of previous attacks, enzyme levels may not rise because the pancreas was damaged and there
are few cells left to release enzymes. Other diagnostic laboratory tests include complete blood count, metabolic profile, lipid profile, and liver function tests.

**COMPLICATIONS, TREATMENTS, AND CARE**

Potential complications of acute pancreatitis are deficient fluid volume, impaired gas exchange, acute pain, risk for infection, and imbalanced nutrition (less than body requirements). Patients with necrotizing pancreatitis may require invasive hemodynamic monitoring, cardio-pulmonary and ventilatory support, and dialysis. Nursing goals include managing symptoms, assessing for complications, and providing family-centered care.

Inflammation leads to significant fluid deletion, and more than 6 L of fluid can be lost into the retroperitoneal space and abdominal cavity (Bentrem & Joehl, 2003). Therefore, fluid resuscitation is the single most important intervention. Hypovolemia can lead to significant renal and cardiac complications. Hemodilution may be necessary to help patients with multiple organ failure (Holcomb, 2007). Hemodynamic monitoring may be necessary to manage symptoms of hypovolemic shock and vasodilation. Shock and vasodilation lead to increased vascular permeability and the complication of thrombus formation and emboli. Emboli may develop from changes in blood composition, immobility, and hyperlipidemia. Triglyceride-lowering medications can be administered to decrease the risk of fatty emboli developing (Holcomb).

Pulmonary complications develop when pancreatic fluid leaks into the pleural space, causing pleural effusions (Holcomb, 2007). This condition puts the patient at risk for pneumonia, atelectasis, pulmonary emboli, and acute respiratory distress symptoms. Intubation may be necessary to decrease respiratory effort and provide support to organ systems (Wroblewski et al., 1999). Nursing interventions to prevent pulmonary complications in intubated patients are following the standards of care for ventilator-associated pneumonia.

Intense pain of acute pancreatitis causes an increase in the release of pancreatic enzymes, requiring absolute treatment (Burruss & Holz, 2005). Meperidine, which was thought to lessen the risk of spasm at the sphincter of Oddi, is no longer supported over other opioids to manage acute pancreatitis pain (Holcomb, 2007). Nursing interventions include pain assessment and administration of intravenous opioids as appropriate.

Administering prophylactic antibiotics has been an area of clinical debate, but it appears to be helpful in appropriately identified cases. Broad-spectrum antibiotics with the ability to penetrate pancreatic tissue are an ideal choice (Pitchumoni, Patel, & Shah, 2005).

Due to increased metabolic needs and nothing-by-mouth status, maintaining adequate nutrition is necessary. Total parenteral nutrition administered through a central line is used for patients with acute necrotizing pancreatitis, although this is not without the risk of infection or sepsis. Recent studies support enteral feedings with nothing by mouth status as long as the nasoenteric tube is advanced into the jejunum to prevent pancreatic enzyme release. Enteral feeding is preferred over parenteral because it is less costly, maintains gut integrity, and enhances immune system functioning (Holcomb, 2007). Insulin production is impaired from the autodigestion process, which results in a damaged endocrine system. This causes hyperglycemia and requires insulin control.

Initially, nonoperative management for severe acute pancreatitis is preferred. If a patient is not responding to medical management or is experiencing acute abdomen, sepsis, or shock, surgical intervention will be necessary and often requires more than one procedure (Wroblewski et al., 1999).

**SUMMARY**

Acute pancreatitis is a complicated disease process that presents many challenges for nurses. Despite treatment, severe acute necrotizing pancreatitis has a high mortality rate. Recognizing signs and symptoms, being knowledgeable of assessment criteria, and providing supportive care can assist the nurse in improving patient outcomes. It is important for nursing to promote patient and family understanding of this disease and prepare both for a potentially long recovery process.

**REFERENCES**


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