Update on Acute Pancreatitis.

by Barbara S. Aronson

Acute pancreatitis is a complex, life-threatening disease that requires diligent and comprehensive medical and nursing care. Recent research and expert panels consensus statements provide new direction with regard to the causes, diagnosis, prognostic indicators, treatment, and nursing care of patients with acute pancreatitis.

Objectives

This independent study offering is designed for nurses and other health care professionals who care for and educate adult patients regarding acute pancreatitis. The multiple choice examination that follows is designed to test your achievement of the following educational objectives. After studying this offering, you will be able to:

1. Describe the pathophysiology and etiology of acute pancreatitis.

2. Discuss diagnostic and prognostic indicators for acute pancreatitis.

3. Outline key components of the current clinical practice guidelines for the treatment of acute pancreatitis and subsequent nursing care.

4. Identify the major complications of acute pancreatitis and related preventative nursing practices.

Acute pancreatitis is a complex, life-threatening disease that has many causes, few effective treatments, numerous serious complications, and an often unpredictable course. The disease may range from a mild, self-limiting inflammatory process to extensive necrosis and multi-organ failure.

Despite recent advances in diagnosis and treatment, acute pancreatitis continues to be a very serious illness with an overall mortality of 5% to 10% (Banks, 1997), and the incidence of this disease appears to be rising (Steinberg & Tenner, 1994). More than 108,000 hospitalizations for pancreatitis in the United States (National Hospital Discharge Survey, 1989) and 2,251 deaths (National Center for Health Statistics, 1987) occur annually. Thus, acute pancreatitis is a significant management problem for adult-health nurses.

In 1997, an expert panel released important acute pancreatitis practice guidelines which incorporate the latest research findings available. The purpose of this review is to share these guidelines and update medical/surgical nurses on the most recent thinking regarding the causes, diagnosis, prognostic indicators, treatment, and nursing care of patients with acute pancreatitis.

Defining Pancreatitis and Recommendations for Care

An improved clinically based classification system for acute pancreatitis and new definitions for the complications associated with the disease were ratified in 1992 by a panel of 40 international experts (Banks, 1997; Bradley, 1993). The panel recommended standard terminology which established the foundation for comparing and consolidating research data. This in turn paved the way for developing clinical guidelines.

Consistent with the use of these expert recommendations, pancreatitis is best defined as an acute inflammatory process of the pancreas that may also involve peripancreatic tissues and/or remote organ systems. Pancreatitis is considered severe if accompanied by the presence of either organ failure and/or local complications such as abscess, pseudocyst, or especially pancreatic necrosis. There are two main types of pancreatitis: necrotizing pancreatitis (20% of patients have this type) and interstitial pancreatitis (80%). People with mild pancreatitis usually have interstitial disease. Those who develop necrotizing pancreatitis have some areas of nonviable parenchyma in their pancreas and also usually have necrosis in the peripancreatic fat area. This necrosis can be classified as either sterile or infected.

In both interstitial and necrotizing pancreatitis fluid commonly extravasates from the pancreas and collects in the space surrounding it. This fluid usually disappears on its own without intervention. In contrast, a pancreatic pseudocyst is a collection of pancreatic juice enclosed by a nonepithelialized wall which usually occurs at least 4 weeks after acute pancreatitis or trauma. Most pseudocysts are sterile, although some become infected, usually through transportation of bacteria from the GI tract. These are now referred to as pancreatic abscesses. A pancreatic abscess is defined as a circumscribed intra-abdominal collection of pus around the pancreas. Abscesses may occur as a result of an infected pseudocyst or when an area of pancreatic necrosis liquefies and becomes infected. Abscesses of either origin usually do not occur until 4 to 6 weeks after the initial onset of pancreatitis.

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Pathophysiology and Etiology

The pathophysiology of pancreatitis remains poorly understood. Regardless of the cause, the process of premature activation of pancreatic enzymes is characteristic, causing varying degrees of inflammation and tissue destruction. It is believed that toxic materials, including pancreatic enzymes, and other toxic substances are released by the pancreas and extravasate into the retroperitoneal spaces, lesser sac, and the peritoneal cavity. The materials cause chemical irritation and help to contribute to third space losses and hypovolemia. Sometimes these toxic substances reach the blood stream and lymphatic pathways and cause organ failure and shock. It is unknown what factors contribute to the intensity of the inflammatory response, although recently leukocytes and their byproducts, and impairment of the microcirculation of the pancreas have been implicated. Necrosis is thought to be also the result of a disruption of the microcirculation of the pancreas and the direct effect of pancreatic enzymes on pancreatic parenchyma (Banks, 1994b, 1997).

In the United States, gallstones are the most common cause of acute pancreatitis (45% of the cases) with alcohol being the second most common cause (35% of the cases) (see Table 1). After gallstones and alcohol abuse, miscellaneous causes account for 10% of the cases. Another 10% of the cases are idiopathic. Outside of the United States, there are numerous etiologic agents.

Table 1.

Causes of Acute Pancreatitis in Adults

Obstructive Causes

* Choledocholithiasis (45%)

* Pancreatic or pancreatic ductal tumors

* Hypersensitive sphincter of Oddi

* Worms or foreign bodies

* Pancreas divisum

* Choledochocele

* Periampullary duodenal diverticula

Toxins

* Ethyl alcohol (35%)

* Methyl alcohol

* Scorpion venom

* Insecticides

* Drugs

Trauma

* Accidental

* Postoperative trauma

* ERCP

* Sphincterotomy

* Manometry of sphincter of Oddi

Metabolic Abnormalities

* Hypertriglyceridemia

* Hypercalcemia

Infection

* Parasitic

* Viral

* Bacterial

Vascular Abnormalities

* Ischemia-post cardiopulmonary bypass

* Emboli

* Vasculitis

Miscellaneous Conditions

* Penetrating peptic ulcer

* Crohn's disease

* Hypothermia

Idiopathic causes (10%) (All the rest =10%)

Adapted from: Steinberg, W., & Tenner, S. (1994). Acute pancreatitis. New England Journal of Medicine, 330(17), 1199.

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Gallstones are the most common obstructive cause of pancreatitis. Women between the ages of 50 to 60 years of age are at highest risk for gallstones. Other obstructive causes for pancreatitis include pancreatic or pancreatic ductal tumors, or a hypersensitive sphincter of Oddi. Any mass that obstructs the pancreatic ducts can result in pancreatitis, presumably by causing reflux of bile into the pancreatic ductal tree, damaging acinar cells (Steinberg & Tenner, 1994).

It is well established that it takes many years of alcohol abuse for pancreatitis to develop. However, since only 5% of all persons who do abuse alcohol subsequently develop pancreatitis, predicting the disease in this population is difficult. Some experts contend that it takes an average of 9 years of heavy alcohol exposure before clinical pancreatitis is seen (Meier, 1995). Researchers believe that alcohol somehow activates pancreatic enzymes in the pancreatic bed. These enzymes generate an inflammatory response that leads to the histologic changes. Other factors also appear to be involved but research has failed to explain the mechanisms causing alcohol-induced pancreatitis (Meier, 1995).

Although alcohol is the most common toxin known to cause pancreatitis, scorpion venom, insecticides, and certain drugs have been identified as inducing the disease. The three drugs with the highest probability for causing pancreatitis are azathioprine, mercaptopurine, and didanosine, although other drugs may also cause injury to the pancreas immediately or months after the exposure. Blunt trauma to the abdomen, with disruption of the pancreatic ductal system, can cause pancreatitis. One to ten percent of persons undergoing ERCP (endoscopic retrograde cholangiopancreatography) may develop pancreatitis, and this incidence is rising due to the availability and increased uses for ERCP (Steinberg & Tenner, 1994).

Pancreatitis may also develop after surgery, especially after the use of cardiopulmonary bypass. Hypertriglyceridemia (with triglyceride levels [is greater than] 1,000 mg per deciliter) and hypercalcemia (rarely) have also been associated with pancreatitis. Viral, bacterial, and parasitic infections may also cause pancreatitis. Patients with AIDS develop pancreatitis at a much higher rate than the general population, and this is thought to be due to an underlying opportunistic viral infection of the pancreas, a neoplasm, or from antiretroviral agents (Maxson, Greenfield, & Turner, 1992). Obesity may be a risk factor in the development of severe pancreatitis (Banks, 1994a & b).

Diagnosis

Almost all patients with acute pancreatitis experience abdominal pain, usually localized to the epigastrium or upper abdomen, and radiating to the back in about 50% of the cases. The pain usually has a quick onset, and is often described as deep, piercing, and unbearable, and often continues without relief for many hours. The pain is frequently aggravated by food, alcohol, vomiting, and resting in the supine position, although the fetal position or sitting may help to relieve his pain. The pain is frequently accompanied by nausea, vomiting, tachycardia, and fever (usually less than 102 F). The patient's abdomen may be distended and tender to palpation and guarding is common. Bowel sounds are usually hypoactive. Rarely, flank ecchymosis (Grey Turner's sign) or periumbilical ecchymosis (Cullen's sign) may be seen. If biliary tree obstruction is present, the patient may be jaundiced.

Serum amylase and serum lipase levels are elevated to three times the upper limits of normal in patients. Serum lipase testing is preferable, since its elevation is more specific than amylase which may be elevated for other disorders that cause abdominal pain. Lipase elevations typically last longer than amylase elevations, so it is especially useful when diagnosing a patient who is seen several days after the illness begins. Lipase levels also have the advantage of being high in patients with both obstructive and alcoholic types. The height of the serum amylase or lipase levels does not correlate to the severity of the pancreatitis, and once the diagnosis has been made, daily testing is not usually necessary. If a patient has a history consistent with pancreatitis (the typical signs and symptoms and a highly elevated serum lipase level), the diagnosis of pancreatitis is an almost certainty ("One Quick Test," 1994).

Patients with gallstone pancreatitis typically have an ALT (alanine amino transferase) of [is greater than] 80 units per 100 ml and this test is often helpful when differentiating alcoholic from gallstone pancreatitis. In response to the inflammatory process, WBC counts are usually elevated in most people with pancreatitis. Liver enzymes may be elevated in patients with alcoholic liver disease.

Diagnostic Tests

Dynamic contrast-enhanced CT scan is the method of choice in visualizing the pancreas, as well as determining the severity of pancreatitis and identifying complications. CT scans should be performed on patients who have severe pancreatitis and/or organ failure, and this imaging technique is especially useful in distinguishing interstitial from necrotizing pancreatitis. Although some patients with mild pancreatitis may have normal CT scans, the vast majority of patients with moderate or severe pancreatitis will have abnormal CT scans. If renal failure is present or

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the patient is allergic to the intravenous contrast, a nonenhanced CT scan can detect some abnormalities such as fluid collections, but cannot differentiate interstitial from necrotizing pancreatitis.

Abdominal ultrasound is recommended within the first 48 hours of hospitalization, especially if obstructive pancreatitis is suspected. Ultrasound helps to diagnose gallstone pancreatitis because it provides visualization of the biliary tree and gallstones, and/or dilatation or obstruction of the common bile duct. An ERCP with sphincterotomy is used to locate and remove gallstones in the common bile duct in patients with gallstone pancreatitis. Early stone extraction (within 24 hours) appears to lower the risk of biliary sepsis and mortality (Banks, 1994b, 1997; Steinberg 1994).

Early identification of patients with acute pancreatitis who have a high risk of developing complications is of paramount importance to their management (Dominquez-Munoz et al., 1993.) Several objective scoring systems have been developed that are useful in determining the severity of pancreatitis. Ranson's 11 prognostic signs provide an effective indicator of the severity of the disease, the first five criteria are used on admission and the last six within 48 hours of admission. A score of [is greater than] 3 on Ranson's signs (see Table 2) during the first 2 days of admission are usually indicative of a severe course of the disease. Mortality approaches 50% when there are six or more Ranson's criteria present.

Table 2.

Ranson's Criteria of Severity at Admission

* Age > 55 years

* WBC > 16,000

* Glucose > 350 IU/L

* LDH > 350 U/L

* AST > 250 U/L

During Initial 48 Hours

* Hct decrease of > 10 vol%

* BUN increase of > 5 mg/dl

* Ca < 8 mg/dl

* Pa[O.sub.2] < 60 mmHg

* Base deficit > 4mEq/L

* Fluid sequestration > 6L

Adapted from: Steinberg, W., & Tenner, S. (1994). Acute pancreatitis. New England Journal of Medicine, 330(17), 1199.

APACHE-II (a simplified Acute Physiology and Chronic Health Evaluation) is another objective measure that can be used to determine severity of disease, and is advantageous because it can be measured at admission, and daily afterwards. When APACHE-II scores are [is less than] 8 on admission the patient usually survives while subsequent increasing scores during hospitalization increase mortality and morbidity.

Although the presence of organ failure is the most reliable indicator of severity of pancreatitis, evidence of significant third-space fluid losses, and the severity of local complications such as necrosis, pseudocyst, and abscess are also indicators of a severe course of the disease. The signs of severe pancreatitis often appear shortly after the onset of the disease and a delayed progression from mild to severe pancreatitis is rare. It is believed that the majority of deaths in acute pancreatitis occur among patients with infected necrosis (abscess) (Banks 1994b, 1997; Bradley, 1993).

Treatment and Nursing Care

Treatment for pancreatitis is generally supportive and dependent on the severity of the disease (see Figure 1). Supportive care includes close monitoring, eliminating of oral intake, effective pain control, fluid replacement, nutritional care, correction of glucose and electrolyte abnormalities, and psychosocial support. In mild pancreatitis, this can usually be accomplished on a general medical/surgical unit, although nurses there need to be vigilant for evidence of a deterioration in the patient's condition, emerging complications, or signs of organ failure (see Table 3). Patients who exhibit signs early in their hospitalizations may develop severe pancreatitis and are usually transferred to an ICU setting for close monitoring and supportive care.

Figure 1.

About the Pancreas

The pancreas is an organ in the abdomen that lies close to the curve of the stomach and the small intestine. It is about six inches long and one inch thick. One part of the pancreas makes insulin and glucagon (the hormones that control blood sugar), and the other part makes enzymes

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that help the small intestine break down the fats and proteins in foods.

What Is Acute Pancreatitis?

Acute pancreatitis is a sudden inflammation of the pancreas and the tissues surrounding it caused by the leakage of enzymes from the pancreas. These enzymes destroy the cells of the pancreas and cause inflammation and fluid collection around the organ. Sometimes these toxic fluids travel to other organs and cause damage there, too.

What Causes Pancreatitis?

* The most common cause in the United States is gallstones that block the passages around the pancreas and gallbladder.

* The second most common cause is alcohol use. It usually takes many years of heavy alcohol use before pancreatitis develops although not every heavy drinker gets pancreatitis.

* Other causes-sometimes certain drugs, infections, surgery, and injury to the abdomen can cause pancreatitis. In other cases, the cause can not be found.

How Is Pancreatitis Diagnosed?

Most patients with acute pancreatitis have moderate to severe pain in their abdomen. Sometimes patients feel pain in their back, too. Most patients will have a fever and nausea and some will vomit. Your doctor will order blood tests or x-rays to be sure that the pain you have is really caused by acute pancreatitis.

* Blood tests -- (called amylase and lipase) are almost always increased when you have acute pancreatitis. Other blood tests (like blood glucose) will help to monitor your overall condition and watch for complications.

* Abdominal CT scan -- this special type of computerized x-ray allows the pancreas and surrounding organs to be seen clearly.

* Abdominal ultrasound -- this test uses sound waves to produce an image on a special screen. This test will show if gallstones caused your pancreatitis.

How Is Pancreatitis Treated?

* Careful monitoring -- your RN will check your vital signs, abdomen and lungs and ask you about your pain and other symptoms frequently

* Pain control -- narcotics are usually given frequently through your intravenous line or through a special pump that you help control. Narcotics are needed until the pain subsides -- for a few days or up a few weeks

* Intravenous fluid -- you will need extra fluids to help replace what you have lost. Your nurse will count your intake (IV fluids) and output (urine, diarrhea, or vomit) every 8 hours. Some patients may need a tube (placed through their nose into their stomach) to control nausea or vomiting.

* Diet -- you will usually not be allowed to eat or drink anything until your inflammation subsides. Sometimes, total parental nutrition is ordered through a special intravenous line. This special formula will supply all of the needed nutrients, vitamins and minerals until you can eat. Most patients will need to follow a low-fat, high-carbohydrate, low-protein diet when they fully recover.

What Can You Do to Help in Your Recovery?

* Let your nurse know if your pain is not relieved -- controlling your pain is an important part of your treatment and recovery. Your nurse can give you some ideas on other ways to get control over your pain. Some patients worry that they will become addicted to the pain medicine. Research has shown that this is not very likely.

* Activity -- although you need to take frequent rests, it is also important to get up and walk. Try to time your activity when your pain medicine is working the best. Coughing and taking deep breaths is important to keep your lungs clean Your nurse will teach you how to do these and other special exercises.

* Many patients benefit from the help of other health care providers: dieticians, physical therapists, social workers, substance abuse counselors or others while they are recovering from acute pancreatitis.

* Your nurse will begin planning for your discharge early and leave you with instructions about your diet, follow up plan and medications. Be sure to ask your nurse or doctor about any special concerns or questions you have about your discharge plan.

Table 3.

Signs of Organ Failure

* Shock-systolic BP < 90 mm Hg

* Renal failure, creatinine > 2 mg/dL

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* Pulmonary insufficiency-PaO < 60 mmHg

* GI bleeding > 500 ml/24 hours

Effective pain management is a priority for patients with pancreatitis, since the pain of pancreatitis is often severe and unrelenting. Effective pain control usually requires narcotic agents via frequent, round the clock intravenous injections or by the patient-controlled anesthesia (PCA) mode. The PCA mode has the advantage of allowing patients more control over pain, and a more uniform drug delivery. Demerol[R] used to be the drug of choice for pancreatitis since morphine was thought to cause spasm of the sphincter of Oddi, but recently this practice has been challenged (Krumberger, 1993; Ruth-Sahad, 1996). According to the Agency for Health Care Policy and Research guidelines for managing acute pain, Demerol should be reserved for very brief courses of acute pain management (U.S. Department of Health and Human Services, 1992). Normeperidine, which is a toxic metabolite of Demerol, acts as a cerebral irritant and may cause dysphoria, mood changes, hallucinations, and seizures. More investigation is needed to determine the best analgesic for patients with pancreatitis.

Nurses should assess a patient's pain frequently, using an objective pain scale, and the effectiveness of the pain regimen. Nonpharmacologic interventions such as guided imagery, and relaxation exercises may be a helpful addition to narcotics for some patients. Patients with pancreatitis are often on narcotic analgesia for a long period of time, up to several weeks. Patients may fear they will become addicted to the narcotics, or become discouraged about their lack of progress in resolving their pain, and need significant nursing support and education to overcome these fears. Inform patients that psychologic dependence and addiction are extremely unlikely to develop after the use of opioids for acute pain (USDHHS, 1992).

Patients generally require vigorous fluid replacement to prevent hypovolemia caused by third-space losses and vomiting, usually with a colloid or Ringer's lactate solution. Aggressive fluid replacement will hopefully limit the development of systemic complications such as shock and renal failure (Banks, 1997). Current research indicates that adequate hydration may also help to prevent pancreatic necrosis by enhancing microcirculation in the pancreas (Banks, 1997). Nurses must carefully monitor intake and output, weight and lab values, and watch for signs of hypovolemia and/or third-space loss. Decreased skin turgor and capillary refill, dry mucous membranes, and thirst are physical assessment indications of hypovolemia. Increases in serum Na, hematocrit, BUN, and urinary Na may indicate hypovolemia. The nurse should be alert for evidence of significant third-space losses. Hemoconcentration (hematocrit [is greater than] 50%), oliguria, azotemia, tachycardia, or hypotension are signs that may indicate a need for more aggressive fluid replacement.

Patients are generally kept NPO to allow the gut to rest and to prevent secretion of pancreatic enzymes. The use of standard treatment regimens such as NG tubes and drugs to reduce gastric secretions may not be of benefit in treating pancreatitis. Controlled trials have shown that nasogastric tubes do not increase pain relief or shorten hospital stays and are appropriate only if patients have severe vomiting, an intestinal ileus, or are at significant risk for aspiration. The use of drugs such as H2 antagonists, which inhibit gastric secretions, are not useful in treating pancreatitis (Banks, 1997). Patients who are NPO need daily mouth assessments and regular oral care.

Patients with pancreatitis are at significant nutritional risk due to a variety of factors. Most patients have a decreased food intake prior to hospitalization due to anorexia and vomiting. Alcoholics especially may be malnourished before developing pancreatitis. In addition, patients with active pancreatitis have increased caloric needs due to inflammation, fever, protein and fluid loss, and possible infection (Noone, 1995).

Nutritional support through total parenteral nutrition (TPN) is required if a patient is NPO for more than a few days (Krumberger, 1993). Lipids are included in the TPN formula unless serum triglycerides are elevated. Nurses should request a nutritional consult early in the disease process before further nutritional depletion occurs. Dietitians will monitor nutritional labs such as albumin, prealbumin, and transferrin on a regular basis to evaluate the effectiveness of the nutritional plan. Serum albumin levels, especially, may be low due to fluid and protein third-space losses. Patients may benefit from a PICC (peripherally inserted central catheter) line, which permits central venous access for TPN and blood drawing without the risk of a subclavian, femoral, or jugular CVP line.

Glucose levels should be monitored every 6 hours when a patient is on TPN. Patients are also at risk for hyperglycemia because of the possible damage of islet cells in the pancreas and may need insulin to combat consistently elevated blood glucose levels. There are no precise guidelines on when oral intake is best resumed. Most physicians consider allowing patients to eat when abdominal pain has subsided, narcotic analgesics are discontinued, active bowel sounds are present, and patients say that they are hungry. Because a high carbohydrate diet is thought to stimulate pancreatic secretions less than a diet high in protein or fat, initially

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patients are placed on small feedings of a moderate to high CHO, low-protein, low-fat diets. Pancreatic enzyme replacement to improve fat absorption may be necessary when oral feedings resume.

Preventing Complications

Patients with pancreatitis can develop a number of grave complications (see Table 4). The most serious pulmonary complication is the development of acute respiratory disease syndrome (ARDS) due to hypovolemia, shock, organ failure, or sepsis. Bilateral infiltrates on X-ray, accompanied by hypoxemia despite high concentrations of oxygen, are hallmarks of ARDS (Noone, 1995). Acute respiratory failure has been implicated in 60% of deaths from acute pancreatitis during the first 7 days of illness (Krumberger, 1993). Atelectasis and pneumonia may develop from an ineffective cough due to abdominal pain and immobility. Respiratory assessment may reveal decreased breath sounds, wheezes, or crackles. Encouraging early ambulation, coughing, and deep breathing, changing the patient's position every 2 hours, and incentive spirometry may prevent this complication. Fevers above 102 F should be reported as patients are at risk for infection from a variety of sources.

Table 4.

Complications of Acute Pancreatitis

Organ Failure

* Hypovolemic shock

* Sepsis

* Coagulopathy

* Respiratory failure (ARDS)

* Acute renal failure

Necrosis

* Sterile

* Infected (Abscess)

Pseudocyst

* Sterile

* Infected (Abscess)

Electrolyte Abnormalities

* Hyperglycemia

* Hypocalcemia

* Hypoaluminemia

* Hypokalemia

Miscellaneous

* Atelectasis

* Pleural effusion

* Pneumonia

* Deep vein thrombosis

Nurses should watch for any symptoms of respiratory distress and monitor pulse oximetry every 4 hours. A decreasing oxygen saturation level may indicate the need for oxygen therapy and a deterioration in the patient's respiratory status. Patients are at risk for pulmonary embolism due to the hypercoagulability and microcirculatory congestion, and for pleural effusions as pancreatic exudate crosses the diaphragm and enters the pleural cavity (Smith, 1991). Nurses should also be alert for pain, swelling, or redness in the calf or thigh, which are characteristic signs of a deep vein thrombosis. Pneumatic stockings and early ambulation may help decrease this risk.

The patient with pancreatitis is at risk for potentially serious cardiovascular and renal complications. Tachycardia, hypotension, profound decreased cardiac output, and increased peripheral vascular resistance are common occurrences in patients with severe pancreatitis and impending organ failure (Krumberger, 1993). Nurses should monitor vital signs frequently and watch for decreasing blood pressure and an increasing pulse rate -- early signs of shock. It is also crucial to assess urinary output regularly, and notify the physician when urinary output is less than 30 cc/hr. Patients are at risk for decreased renal perfusion and acute renal failure due to hypovolemia and hypotension. The glomerular filtration rate drops with hypovolemia, and induces the release of renin, which constricts blood vessels and elevates aldosterone levels, decreasing renal perfusion. Antidiuretic hormone is also released in response to hypovolemia, causing further sodium and water retention. In addition, hypercoagulability can impair renal perfusion and worsen renal vascular resistance, which in turn can further lead to renal failure.

Electrolyte imbalances can be potentially life threatening

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for patients with pancreatitis and nurses should monitor these values frequently, and administer replacements as needed. Hypokalemia, hypocalcemia, and hypomagnesemia are the most frequently seen electrolyte imbalances, especially among alcoholics. Hypocalcemia (calcium level [is less than] 8.0 mg%) is an electrolyte abnormality that is often associated with severe pancreatitis and persistently low levels increase mortality. Signs of low calcium include neuromuscular irritability (tingling sensations), nausea, vomiting, decreased cardiac output, and laryngeal spasm. Monitor the patient for signs of tetany which include positive Chvostek's and Trousseau's signs. Patients with pancreatitis are at risk for hypokalemia (potassium [is less than] 3.5 mEq/L) due to prolonged vomiting or the loss of potassium in the protein-rich fluid that leaks into the peritoneal cavity. Signs of low potassium include muscle weakness, hypotension, and arrhythmia. On the other hand, expect potassium to be elevated if patient develops acute renal failure.

Changes in mental status seen in patients with pancreatitis may be due to intense pain, impending shock, sleep deprivation, or alcohol withdrawal. Patients will often be anxious and restless, due to the pain and anxiety related to their disease process. Try to schedule activities and treatments in blocks of time to allow patients some uninterrupted time for rest; however, the value of enforced strict bedrest has never been substantiated (Ambrose, 1996). In fact, patients may suffer more from the hazards of immobility then from a gradual increase in planned activity. Include range of motion exercises and early ambulation in the plan of care to decrease muscle wasting and prevent complications from immobility.

When caring for patients with alcoholic pancreatitis be especially alert for signs of alcohol withdrawal such as tachycardia, restlessness, agitation, tremors, anxiety, and diaphoresis. Recognizing the symptoms and obtaining pharmacologic treatment in the early stages of withdrawal may prevent progression to alcohol withdrawal syndrome, which is an acute medical emergency. Patients in active withdrawal will need scheduled sedation with benzodiazepines and close monitoring, as well as effective pain control.

The use of antibiotics is usually reserved for patients with necrotizing pancreatitis and organ failure, or pancreatic abscess. According to recent research, antibiotics do not seem to prevent pancreatic infection (Banks, 1994b, 1997). Patients with necrotizing pancreatitis who improve with supportive care are generally treated medically and monitored closely in an intensive care setting. If patients with necrotizing pancreatitis do not improve, a guided percutaneous aspiration is usually performed to distinguish infected necrosis from sterile necrosis. If a successful aspiration is obtained, a gram stain and culture are obtained. Fever and increased WBC may occur with both infected and noninfected necrosis often making the diagnosis difficult. If infection is documented, surgical debridement is indicated. Patients with sterile necrosis are usually treated medically, although the mortality rate tends to be high. Pancreatic pseudocysts resolve spontaneously 25% to 50% of the time and asymptomatic pseudocysts usually require no treatment. If pseudocysts are symptomatic, they may be drained surgically, radiologically, or endoscopically.

Psychosocial Support and Discharge Planning

Patients with severe pancreatitis require extensive emotional support due to the often lengthy course of hospitalization, the pain, and the uncertainty about recovery. The patient may express guilt if they recognize drinking caused their illness. Referral to a substance abuse counselor for recovery programs is a necessity. Dietary instruction regarding diet and avoiding caffeine and spices is needed. Discharge planning should begin on admission, as the patient and his family may need resources when finally returning home. Enlist the assistance of a visiting nurse to help monitor the patient's condition and medications and/or physical therapy if loss of mobility and muscle strength are an issue. Financial assistance is often an issue, particularly if the patient is uninsured or poorly covered for a lengthy hospitalization.

Patients will need teaching about their disease process and signs and symptoms to report to health care providers that may indicate a reoccurrence or complications. Education about preventative health practices such as smoking cessation, nutrition, exercise, and routine followup care are important. In addition, the patient may need specialized teaching if diabetes has developed as a result of the illness, including insulin injection techniques and monitoring blood glucose levels. Remember to include individualized medication instruction if the patient goes home on pancreatic enzymes or antibiotics.

Acute pancreatitis, while usually very serious and even life threatening, is increasingly better understood. Adult health nurses must be aware of the 1997 practice guidelines that direct the diagnosis and treatment of the disease. Patients with acute pancreatitis often have complex needs and require significant teaching and psychosocial support. They present medical-surgical nurses with an important opportunity to provide holistic care that can significantly improve patients' quality of life.

References

Ambrose, M. (1996, April). Pancreatitis: Managing a

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flare-up. Nursing 96, 33-39.

Banks, P. (1997). Practice guidelines in acute pancreatitis. American Journal of Gastroenterology, 92(3), 377-386.

Banks, P. (1994a). Acute pancreatitis: Conservative management. Digestive Surgery, 11, 220-225.

Banks, P. (1994b). Acute pancreatitis: Medical and surgical management. American Journal of Gastroenterology, 89(8), S78-S85.

Bradley, E. (1993). A clinically based classification system for acute pancreatitis. Archives of Surgery, 128, 586-590.

Dominquez-Munoz, J., Carballo, F., Garcia, M., Diego, J., Campos, R., Yanguela, J., & Morena, J. (1993). Evaluation of the clinical usefulness of Apache II and Saps systems in the initial prognostic classification of acute pancreatitis: A multicenter study. Pancreas, 8(6), 682-686.

Krumberger, J. (1993). Acute pancreatitis. Critical Care Nursing Clinics of North America, 5(1), 185-203.

Maxson, C., Greenfield, S., & Turner, J. (1992). Acute pancreatitis as a common complication of 2,3-dideoxyinosine therapy in the acquired immunodeficiency syndrome. American Journal of Gastroenterology, 87(6), 708-713.

Meier, P. (1995). Editorials: Who gets and what causes pancreatitis? Journal of Laboratory Clinical Medicine, 125, 298-300.

National Center for Health Statistics, Vital statistics of the United States, Vol. II. Part A. Mortality. (1987). (DHHS publication #PHS 87-1101). Washington, DC: U.S. Government Printing Office.

National Hospital Discharge Survey. (1989). Detailed diagnoses and procedures. Vital Health Stats, 13(100), 1-304.

Noone, J. (1995, August). Acute pancreatitis: An Orem approach to nursing assessment and care. Critical Care Nurse, 27-35.

One quick test for acute pancreatitis. (1994, May). Emergency Medicine, 119.

Ruth-Sahad, L. (1996). Clinical snapshot: Acute pancreatitis. American Journal of Nursing, 96(6), 38.

Smith, A. (1991, September). When the pancreas self-destructs. American Journal of Nursing, 39-48.

Steinberg, W., & Tenner, S. (1994, April). Acute pancreatitis. The New England Journal of Medicine, 1198-1210.

United States Department of Health and Human Services, Agency for Health Care Policy and Research. (1992). Quick Reference Guide for Clinicians (#1), Acute Pain Management in Operative Procedures. Rockville, MD: USDHHS.

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