Care of a Client with Pancreatic diseases

Understanding pancreatitis and nursing management of complications.

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Introduction

Inflammation of the gland (pancreatitis) is the most significant benign condition of the organ, and may be acute or chronic. Many patients will recover spontaneously regardless of the cause or the treatment, while others experience a fulminant course with multi-organ failure (shock, respiratory failure, renal failure), and death. Treatment dependent on the cause of the symptoms, and in most cases is largely supportive in nature. Disorders affecting the pancreas include pancreatitis,

- Pancreatitis
  - Acute pancreatitis
  - Chronic Pancreatitis
- Pancreatic lesion or cancer
- Diabetes

This review will discuss management of acute and chronic pancreatitis.

Learning objectives

At the end of this review the reader should be able to

- Describe the functions of the pancreas.
- Identify the causes of acute pancreatitis.
- State the difference between acute and chronic pancreatitis.
- Discuss the diagnostic procedures for pancreatitis.
- Discuss the management of pancreatitis.
Functions of the Pancreas

The pancreas, an elongated ‘fish’ shaped organ situated inferiorly and posteriorly to the stomach on the left side of the abdominal cavity. The tail of the pancreas and spleen are in the left upper quadrant of the abdomen and the head of the pancreas is in the right upper quadrant just to the right of the midline. The head of the pancreas lies in the loop of the duodenum as it exits the stomach. The pancreatic duct runs along the length of the pancreas and connect with the common bile duct near the head of the pancreas. The pancreas has important endocrine and exocrine functions.

Exocrine functions

The exocrine function of the pancreas is carried out by the acinar tissue. The acinar tissue is composed of tiny grapelike clusters of cells that produce the pancreatic fluid. The pancreas pancreatic fluid consist of water, mineral salts and digestive enzymes; amylolytic enzymes (amylase), lipolytic enzymes (lipase, phospholipase and cholesterol esterase), and proteolytic enzymes (trypsin, chymotrypsin). These enzymes assist in the breakdown of starch, fats, and proteins during the digestive process. The enzymes trypsin and chymotrypsin are in their inactive forms (Trypsinogen and chymotrypsinogen) until they enter the small intestine where they are activated by enterokinas (enteropeptidase) released in the microvilli of the duodenum. It is very important that these enzymes be released in
their inactive forms and be only activated upon arrival to the duodenum, otherwise they would digest the pancreas.

**Endocrine function**

The endocrine function of the pancreas is carried out by cluster of specialized cells scattered throughout the pancreas called the *Islet of Langerhans*. The Islet of Langerhans contain alpha, beta, delta, and PP cells. The beta cells produce somatostatin, which inhibits the release of insulin and glucagon. The endocrine portion of the pancreas controls the homeostasis of glucose in the bloodstream. The pancreas produces 2 antagonistic hormones to control blood sugar: glucagon and insulin.

The alpha cells of the pancreas produce glucagon. Glucagon raises blood glucose levels by stimulating the liver to metabolize glycogen into glucose molecules and to release glucose into the blood. Glucagon also stimulates adipose tissue to metabolize triglycerides into glucose and to release glucose into the blood.

Insulin is produced by the beta cells of the islets of Langerhans in the pancreas. Insulin lowers blood glucose levels after a meal by stimulating the absorption of glucose by liver, muscle, and adipose tissues. Insulin triggers the formation of glycogen in the muscles and liver and triglycerides in adipose to store the absorbed glucose.

**What is Pancreatitis?**

**Acute pancreatitis**

Acute pancreatitis is an acute inflammatory process of the pancreas that may also involve peripancreatic tissues and/or remote organ systems. This results largely from an auto-digestive process within the pancreas leading to pancreatic parenchymal cell destruction. Characterized clinically by abdominal pain and elevated levels of pancreatic enzymes in the blood.

The diagnosis of acute pancreatitis requires two of the following three features:

1) abdominal pain consistent with acute pancreatitis (acute onset of a persistent, severe, epigastric pain often radiating to the back);
2) serum lipase activity (or amylase activity) at least three times greater than the upper limit of normal; and
3) Characteristic findings of acute pancreatitis on contrast-enhanced computed tomography (CECT) and
less commonly magnetic resonance imaging (MRI) or transabdominal ultrasonography.

**Types of acute pancreatitis**

Acute pancreatitis can be subdivided into two types: interstitial edematous pancreatitis and necrotizing pancreatitis.

**Interstitial edematous pancreatitis**
Acute inflammation of the pancreatic parenchyma and peripancreatic tissues, but without recognizable tissue necrosis. The clinical symptoms of interstitial edematous pancreatitis usually resolve within the first week.

**Necrotizing pancreatitis**
Inflammation associated with pancreatic parenchymal necrosis and/or peripancreatic necrosis. About 5–10% of patients develop necrosis of the pancreatic parenchyma, the peripancreatic tissue or both. The impairment of pancreatic perfusion and signs of peripancreatic necrosis evolve over several days.

**Etiology of acute pancreatitis**
There are many possible underlying causes of acute pancreatitis, but biliary tract disease and alcoholism account for ≥ 80% of acute pancreatitis cases in the United States.

- **Gallstone pancreatitis**: obstruction of the sphincter of Oddi by a gallstone or microlithiasis (sludge).
- **Alcoholic pancreatitis**: Alcohol is a common cause of acute pancreatitis. Alcoholic pancreatitis is more common in individuals who have a long history of alcohol abuse.
- **Drug-induced pancreatitis**: A number of drugs used to treat medical conditions can trigger acute pancreatitis.
- **Post-ERCP**: Acute pancreatitis develops in about 3 to 5 percent of people who undergo Endoscopic retrograde cholangiopancreatography (ERCP). Most cases of ERCP-induced pancreatitis are mild.
- **Hereditary conditions**: Acute pancreatitis can be caused by hereditary conditions, such as familial hypertriglyceridemia (high blood triglyceride levels) and hereditary pancreatitis. These conditions usually occur in children and young adults.
Unexplained (idiopathic) pancreatitis. No underlying cause can be identified in about 20 percent of people with acute pancreatitis.

Pathophysiology of pancreatitis

The exact mechanism that triggers auto digestion is not known. Pancreatic enzymes (including trypsin, phospholipase A2, and elastase) become activated within the pancreas. The enzymes can damage tissue and activate the complement system and the inflammatory cascade, producing cytokines. This process causes inflammation, edema, and sometimes necrosis. In mild pancreatitis, inflammation is confined to the pancreas; in severe pancreatitis, there is significant inflammation, with necrosis and hemorrhage of the gland and a systemic inflammatory response; After 5 to 7 days, necrotic pancreatic tissue may become infected by enteric bacteria. Activated enzymes and cytokines that enter the peritoneal cavity cause a chemical burn and third spacing of fluid; those that enter the systemic circulation cause a systemic inflammatory response that can result in acute respiratory distress syndrome and renal failure. The systemic effects are mainly the result of increased capillary permeability and decreased vascular tone, which result from the released cytokines and chemokines. Phospholipase A2 is thought to injure alveolar membranes of the lungs.

In some patients, collections of enzyme-rich pancreatic fluid and tissue debris form in and around the pancreas. The collections resolve spontaneously in some patients; in others the collections become infected or form pseudocysts. Pseudocysts have a fibrous capsule without an epithelial lining. Pseudocysts may hemorrhage, rupture, or become infected. Death during the first several days is usually caused by cardiovascular instability (with refractory shock and renal failure) or respiratory failure (with hypoxemia and at times adult respiratory distress syndrome) (Severe classification). Occasionally, death results from heart failure secondary to an unidentified myocardial depressant factor. Death after the first week is usually caused by multiorgan system failure.

Classification of Acute Pancreatitis

There are two phases of acute pancreatitis: early and late phase.

The early phase usually lasts for the first week is followed by a second later phase which can run a protracted course from weeks to months, systemic
disturbances during the early phase result from the host response to local pancreatic injury. The late phase is characterized by persistence of systemic signs of inflammation or by the presence of local complications. The late phase occurs only in patients with moderately severe or severe acute pancreatitis. Persistent organ failure is the main determinant of severity.

Severity of acute pancreatitis is divided into the following:

- **Mild acute pancreatitis**: Mild acute pancreatitis, the most common form, has no organ failure, local or systemic complications and usually resolves in the first week.

- **Moderately severe acute pancreatitis**: Moderately severe acute pancreatitis is defined by the presence of transient organ failure (resolves within 48 hours), local complications or exacerbation of co-morbid disease without persistent organ failure (>48 hours).

- **Severe acute pancreatitis**: Severe acute pancreatitis is defined by persistent organ failure, that is, organ failure >48 hours. Local complications are acute peripancreatic fluid collections, pancreatic pseudocyst, and peripancreatic necrosis (sterile or infected), pseudocyst and walled-off necrosis (sterile or infected). Other local complications of acute pancreatitis include gastric outlet dysfunction, splenic and portal vein thrombosis, and colonic necrosis.

**Symptoms of acute pancreatitis**
- Acute onset of steady, boring upper abdominal pain.
- Pain radiates through to the back in some patients.
- Pain usually persists for several days.
- Sitting up and leaning forward may reduce pain, but coughing, vigorous movement, and deep breathing may accentuate it.
- Nausea and vomiting are common. (Pain not relieved by vomiting)
- Patient appears acutely ill and sweaty.
- Tachycardia with pulse rate is usually 100 to 140 beats/min.
- Shallow and rapid respiration
- BP may be transiently high or low, with significant postural hypotension.
- Temperature may be normal or even subnormal at first but may increase to 37.7 to 38.3°C (100 to 101°F) within a few hours.
- Altered level of consciousness.
- Scleral icterus is occasionally present.
- Decreased lung sound (signs of atelectasis)

- Upper abdominal distention caused by gastric distention or displacement of the stomach by a pancreatic inflammatory mass.
- Pancreatic duct disruption may cause ascites (pancreatic ascites).
- Marked abdominal tenderness occurs, most often in the upper abdomen.
- Mild-to-moderate muscular rigidity may be present in the upper abdomen
- Severe peritoneal irritation results in a rigid and boardlike abdomen.
- Bowel sounds may be hypoactive.
- Turner sign (ecchymoses of the flanks) and the Cullen sign (ecchymoses of the umbilical region) indicate extravasation of hemorrhagic exudate.

- Infection in the pancreas or in an adjacent fluid collection should be suspected if the patient has a generally toxic appearance with elevated temperature and WBC count or if deterioration follows an initial period of stabilization.
Diagnostic tests for pancreatic disorders

Pancreatitis is suspected whenever severe abdominal pain occurs, especially in a patient with significant alcohol use or known gallstones. Diagnosis is made by clinical suspicion, serum markers (amylase and lipase), and the absence of other causes for the patient’s symptoms.

ERCP: Endoscopy to examine the gallbladder and pancreas to evaluate obstruction on the common bile duct and to confirm pancreatic disease.

Magnetic resonance cholangiopancreatography (MRCP)

Ultrasonography and CT are not generally done specifically to diagnose pancreatitis but are often used to evaluate acute abdominal pain.

Ultrasonography should be done if gallstone pancreatitis is suspected. Edema of the pancreas may be visualized.

CT with IV contrast is generally done to identify necrosis, fluid collections, or pseudocysts once pancreatitis has been diagnosed (recommended for severe pancreatitis or if a complication occurs).

Percutaneous CT-guided needle aspiration of cysts or areas of fluid collection or necrosis may reveal organisms on Gram stain or culture.

Laboratory Values Seen in Acute Pancreatitis

- **Serum amylase.** An increase of amylase in the blood usually indicates pancreatitis. (3 x higher than normal). (rises within 12 hr, lasts 4 days)

- **Serum lipase.** Sudden (acute) pancreatitis almost always raises the level of lipase in the blood. (Rises slower but lasts up to 2 weeks). Lipase is more specific for pancreatitis.

- A urine dipstick for trypsinogen-2 has sensitivity and specificity of > 90% for acute pancreatitis.

- Elevated White blood cell (WBC) count

- Third-space fluid losses may increase the Hct to as high as 50 to 55%, indicating severe inflammation.

- Hyperglycemia may occur.

- Elevated serum bilirubin in 15 to 25% of patients because pancreatic edema compresses the common bile duct.

- Liver-associated enzymes: alkaline phosphatase, total bilirubin, aspartate aminotransferase (AST), and alanine aminotransferase (ALT)
levels to search for evidence of
gallstone pancreatitis.

An ALT level higher than 150 U/L
suggests gallstone pancreatitis and a
more fulminant disease course.

Complications of acute pancreatitis

**Organ failure** respiratory, cardiovascular
and renal. If organ failure affects more than
one organ system, it is termed multiple
organ failure

**Local complications** Local complications
are acute peripancreatic fluid collections,
pancreatic pseudocyst, and peripancreatic
necrosis (sterile or infected), pseudocyst
and walled-off necrosis (sterile or infected).
Other local complications of acute
pancreatitis include gastric outlet
dysfunction, splenic and portal vein
thrombosis, and colonic necrosis.

**Systemic complications** systemic
complications is defined as the
exacerbation of pre-existing co-morbidity,
such as coronary artery disease or chronic
lung disease, precipitated by the acute
pancreatitis.

Management

**Ranson’s Criteria for Pancreatitis Mortality**

Estimates mortality of patients with
pancreatitis, based on initial and 48-hour
lab values. Helps determine the disposition
of the patient, with a higher score
corresponding to a higher level of care. The
scores are calculated on admission, and at
48 hours, to estimate mortality
from pancreatitis. *(See table 1).*

**48 Hours Into Admission**

- Hct drop \(>10\% \text{ from admission}
- BUN increase \(>5 \text{ mg/dL (}>1.79 \text{ mmol/L) from admissi}
- Ca <8 \text{ mg/dL (}<2 \text{ mmol/L) within 48 hours}
- Arterial pO2 <60 mmHg
- Base deficit (24 - HCO3)  
- Fluid needs > 6L within 48 hours

**On Admission**

- Patient older than 55 years
- WBC count higher than 16,000/\mu L
- Blood glucose level higher than 200 mg/dL
- Serum LDH level higher than 350 IU/L
- AST level higher than 250 IU/L

**Table 1: Ranson’s Criteria**

Treatment

Severe acute
pancreatitis should
be treated in an
ICU, particularly in
patients with
hypotension,
oliguria, Ranson’s
score \(\geq 3\), or
pancreatic necrosis
on CT > 30%.

- Fluid resuscitation. Adequate fluid resuscitation is essential; up to 6 to 8 L/day of fluid containing appropriate electrolytes. Inadequate fluid therapy increases the risk of pancreatic necrosis.

- Fasting, rest the pancreas. Fasting is indicated until acute inflammation subsides (ie, cessation of abdominal tenderness and pain, normalization of serum amylase, return of appetite, feeling better). Fasting can last from a few days in mild pancreatitis to several weeks. In severe cases, TPN should be initiated within the first few days to prevent undernutrition.

- Pain management. Drugs, including adequate analgesia and acid blockers. Pain relief requires parenteral opioids, which should be given in adequate doses.
  - Morphine
  - Antiemetic agents (eg, prochlorperazine 5 to 10 mg IV q 6 h) should be given to alleviate vomiting.
  - An NGT is required only if significant vomiting persists or ileus is present.

- Parenteral H2 blockers or proton pump inhibitors are given.

- ABG is determined as needed;

- Central venous pressure line or Swan-Ganz catheter measurements are determined every 6 h if the patient is hemodynamically unstable or if fluid requirements are unclear.
  - CBC, platelet count, coagulation parameters, total protein with albumin, BUN, creatinine, Ca, and Mg are measured daily.

- Hypoxemia is treated with humidified O2 via mask or nasal prongs. If hypoxemia persists or adult respiratory distress syndrome develops, assisted ventilation may be required.

- Glucose > 170 to 200 mg/dL (9.4 to 11.1 mmol/L) should be treated cautiously with sc or IV insulin and carefully.

- Hypocalcemia generally is not treated unless neuromuscular irritability occurs; 10 to 20 mL of 10% Ca gluconate in 1 L of IV fluid is given over 4 to 6 h.
F Chronic alcoholics and patients with documented hypomagnesemia should receive Mg sulfate 1 g/L of replacement fluid for a total of 2 to 4 g, or until levels normalize. Serum Mg levels are monitored and IV Mg is given cautiously if renal failure occurs. Renal failure may require dialysis.

F Management of infection.
Antibiotics for pancreatic necrosis. Antibiotic prophylaxis with imipenem can prevent infection of sterile pancreatic necrosis.

F Drainage of infected pseudocysts or areas of necrosis. Infected areas of pancreatic necrosis require surgical debridement, but infected fluid collections outside the pancreas may be drained percutaneously. A pseudocyst that is expanding rapidly, infected, bleeding, or likely to rupture requires drainage.

F Surgical intervention during the first several days is justified for severe blunt or penetrating trauma or uncontrolled biliary sepsis. ERCP with sphincterotomy and stone removal is indicated for patients with gallstone pancreatitis who do not improve after 24 h of treatment.

Nursing management of acute pancreatitis

Patient admitted to ICU if needed.
Vital signs and urine output are monitored hourly;
Hct, glucose, and electrolytes) are reassessed every 8 hours.

Rest the pancreas

- NPO – no food until pain free, no nausea and vomiting.
- Administer antiemetic as needed
- Nasogastric tube – gastric decompression
- Total parenteral nutrition (TPN) or jejunal feedings (less risk of hyperglycemia)
- When diet is resumed. bland, low-fat diet with no stimulants (caffeine); small, frequent meals
- No alcohol consumption
- No smoking
- Limit stress

Pain management

- Position the client for comfort (fetal, side-lying, the head of the bed elevated, sitting up or leaning forward).
- Administer analgesics and other medications as prescribed.

Monitor blood glucose levels and provide insulin as needed (potential for hyperglycemia).
Monitor hydration levels (orthostatic blood pressure, intake and output, laboratory values).

Provide IV fluids and electrolyte replacement as prescribed.

**Medications management**

Pain management

- Opioid analgesics, morphine sulfate, hydromorphone, fentanyl.
  - Given for acute pain.
  - Nursing Considerations
    - Large doses of intravenous opioids are often needed for pain management.
    - Meperidin (Demerol) is discouraged in older adult clients due to the risk of seizures.
- H2-blockers, PPIs, antacids decrease acid stimulation of enzymes and prevents stress ulcers.
- Antibiotics may be used, but are generally indicated for clients with acute necrotizing pancreatitis
- Anticholinergics – dicyclomine (Bentyl)
  - This is given to decrease intestinal motility and the flow of pancreatic enzymes.
- Spasmolytics – papaverine (Pavabid)
  - This medication relaxes smooth muscle.
- Pancreatic enzymes (pancreatin [Donnazyme], pancrelipase [Viokase])
  - Given to help with the digestion of fats and proteins when taken with meals and snacks

**Nutritional therapy for acute pancreatitis**

Nutritional therapy for acute pancreatitis involves reducing any pancreatic stimulation. The client is prescribed to have nothing by mouth (NPO), and a nasogastric tube is inserted to suction gastric contents.

- TPN may be used until oral intake is resumed.
- Provide a bland, high carbohydrate, low protein, and low fat diet as tolerated.
  - Increase caloric intake without pancreatic stimulation.
- Patient should avoid alcohol and spicy foods that stimulate the pancreas.

**Selected Nursing diagnosis**

- Acute pain related to:
  - Distention of the pancreas associated with inflammation and obstruction of pancreatic ducts;
peritoneal irritation associated with escape of activated pancreatic enzymes into the peritoneum.

- Ineffective breathing pattern related to splinting from severe pain, pulmonary infiltrates, pleural effusion, and atelectasis.
- Fluid volume deficit, related to decreased oral intake and excessive loss of fluid associated with vomiting and nasogastric tube drainage.
- Electrolyte imbalance (hypokalemia, hypocalcemia and hypochloremia) related to decreased oral intake and excessive loss of fluid and electrolytes associated with vomiting and nasogastric tube drainage.
- Imbalanced nutrition less than body requirement related to decreased oral intake associated with nausea, pain, prescribed dietary restrictions, and feeling of fullness resulting from abdominal distention.
- Altered comfort: abdominal distention and gas pain related to accumulation of gas and/or fluid in the gastrointestinal tract.
- Impaired skin integrity.
- Risk for infection: sepsis related to release of bacteria into the blood associated with presence of infected necrotic areas or leakage of infected pseudocysts or abscesses.

**Chronic Pancreatitis**

Chronic pancreatitis is persistent inflammation of the pancreas that results in permanent structural damage with fibrosis and ductal strictures, followed by a decline in exocrine and endocrine function. It can occur as the result of chronic alcohol abuse but may be idiopathic. Initial symptoms are recurrent attacks of pain. Later in the disease, some patients develop malabsorption and glucose intolerance.

**Causes Chronic Pancreatitis**

Alcohol abuse, idiopathic, hereditary pancreatitis, autoimmune pancreatitis, hyperparathyroidism, and obstruction of the main pancreatic duct caused by stenosis, stones, or cancer.

**Symptoms and Signs**

- Episodic abdominal pain. Pain is epigastric, severe, and may last many hours or several days.
- About 10 to 15% have no pain and present with malabsorption.
- Episodes typically subside spontaneously after 6 to 10 yr as the acinar cells that secrete pancreatic
digestive enzymes are progressively destroyed.

- Steatorrhea.
- Symptoms of glucose intolerance

Diagnosis

Diagnosis can be difficult because amylase and lipase levels are frequently normal because of significant loss of pancreatic function. In a patient with a typical history of alcohol abuse and recurrent episodes of acute pancreatitis, detection of pancreatic calcification on plain x-ray of the abdomen may be sufficient.

- Abdominal CT: CT can show calcifications and other pancreatic abnormalities (e.g., pseudocyst or dilated ducts)
- MRCP can show masses in the pancreas as well as provide more optimal visualization of ductal changes consistent with chronic pancreatitis.
- ERCP, endoscopic ultrasonography, and secretin pancreatic function testing.

Treatment

- A relapse requires treatment similar to acute pancreatitis with fasting, IV fluids, and analgesics.
- Avoid alcohol and consume a low-fat (< 25 g/day) diet (to reduce secretion of pancreatic enzymes).
- An H2 blocker or proton pump inhibitor may reduce acid-stimulated release of secretin, thereby decreasing the flow of pancreatic secretions.
- Pancreatic enzyme supplementation may reduce chronic pain by inhibiting the release of cholecystokinin, thereby reducing the secretion of pancreatic enzymes.
- Enzymes are also used to treat steatorrhea.
- Pancreatic enzyme supplementation should be taken with meals.
- An H2 blocker or proton pump inhibitor should be given to prevent acid breakdown of the enzymes.
- Supplementation with fat-soluble vitamins (A, D, K) should be given, including vitamin E, which may minimize inflammation.
- Surgical treatment may be effective for pain relief.
- Provide education about taking the enzyme replacement therapy. Encourage patient to complete the form that helps track GI symptoms to help monitor their progress while taking the medication.
Pancreatic Surgery

The Whipple operation (also called a pancreaticoduodenectomy) removal of the head of the pancreas, a portion of the bile duct, the gallbladder, the duodenum, a portion of the stomach, and the lymph nodes near the head of the pancreas. The remaining pancreas is then reconnected to the duodenum so that pancreatic digestive enzymes, bile and stomach contents will flow into the small intestine during digestion.

Pseudocysts can be drained endoscopically.

ERCP with sphincterotomy, stent placement, or dilatation.

Insulin should be given cautiously because the coexisting deficiency of glucagon secretion by α-cells means that the hypoglycemic effects of insulin are unopposed and prolonged hypoglycemia may occur.

Patients are at increased risk of pancreatic cancer. Worsening of symptoms, especially with development of a pancreatic duct stricture, should prompt an evaluation for cancer.

Distal Pancreatectomy

In a distal pancreatectomy, the tail and sometimes body of the pancreas is removed. The most frequent reason for performing this surgery is the presence of a tumor in the body or tail of the pancreas.

- Open distal pancreatectomy and splenectomy
- Spleen-preserving distal pancreatectomy

Total Pancreatectomy

The procedure involves the complete removal of the pancreas, spleen, gallbladder, common bile duct, and a portion of the small bowel and stomach. A patient undergoing total pancreatectomy will be unable to produce pancreatic enzyme required for digestion and insulin release for control blood glucose level.

Selected References


NCLEX STYLE QUESTIONS

Case: JT is a 48 year old, divorced business executive brought to the emergency department by his buddies with a chief complaint of abdominal & back pain and vomiting for 2 days. As you approach him you observe that he is trying to sit up, and is almost in continuous movement on the bed. He is alert and able to answer questions, but refuses to let anyone touch his abdomen or his back. He rates his pain at 10/10. His skin is hot, dry and flushed with decreased turgor and he complains of extreme thirst.

VS: BP 100/60, T–100°F, P–120, R– 28 shallow, O2 sats–90%

Use the information to answer questions 1 and 2.

1. Based on the information in the preceding situation, place the following interventions in priority?
   a. Administer O2 @ 2L/nasal cannula
   b. Administer pain medications
   c. Complete the physical assessment
   d. Initiate IV of Normal Saline at 125 ml/hour

2. Which of these medications ordered for the client will the nurse question?
   a. Prochlorperazine 5 mg IV q 6 hours PRN nausea and vomiting.
   b. Morphine sulfate 5 mg orally every 4 hours PRN pain.
   c. Meperidine 2 mg intravenously every 6 hours PRN pain.
   d. Hydromorphone 2 mg IV 4 hours PRN pain.

3. A patient has acute pancreatitis. The patient has a positive Cullen’s sign. A nurse should prioritize assessment of which lab value?
   a. Serum glucose
   b. Trypsin
   c. Creatinine
   d. Hematocrit
4. A patient has been treated for acute pancreatitis. When the patient is permitted a diet, which diet order is anticipated?

   a. No added salt
   b. Vegetarian
   c. 1800 calorie ADA
   d. Low fat

5. The nurse is caring for a client admitted with acute pancreatitis. Which of these new finding should the nurse report immediately to the physician?

   a. Abdominal pain radiating to the back and B/P of 98/55.
   b. Recurrent nausea and vomiting and dry mucous membrane.
   c. Upper abdominal tenderness on palpation heart rate of 110b/min.
   d. Increasing abdominal pain, new onset fever and leukocytosis.

6. The nurse assesses a client admitted with acute pancreatitis whose chief complaints are numbness and tingling of the extremities and leg cramps. Which of the following will be the best intervention to assist the client?

   a. Notify the physician immediately that client is exhibiting signs of hypocalcemia.
   b. Continue to monitor and record the client’s vital signs and neuro signs.
   c. Perform a complete neuromuscular assessment on the client.
   d. Offer client oral calcium gluconate or calcium carbonate.

7. The nurse has an order to insert NG tube into a client with acute pancreatitis who has persistent vomiting. After insertion of the tube client’s voice was hoarse when asked to speak. Which of these is the most appropriate action by the nurse?
a. Remove the tube it is probably in the trachea
b. Insert air and listen for gurgling in the epigastrium
c. Ask the client to cough and clear her throat
d. Send the client for a chest X-ray to confirm placement.

8. A student nurse observed a nurse during insertion of an NG tube give the client sips of water and asked him to swallow as the tube is advanced into the esophagus. Which statement by the nurse correctly explains the reason for this action to the student nurse?

a. It help lubricate the esophagus for easy advancement of the tube
b. It elevates the uvula and helps distract the

c. The epiglottis covers the laryngeal opening so that the tube enters the esophagus.
d. Swallowing water help the client swallow the tube so that it advances into the esophagus.

9. A client with chronic pancreatitis is ordered a combination of a proton pump inhibitor and pancreatic enzyme. The nurse correctly explains the rational for this order to the client as

a. Proton pump inhibitor prevent stress ulcer in client with pancreatitis.
b. Proton pump inhibitor prevents the breakdown of enzymes by gastric juices.
c. Proton pump inhibitor and an H2 blocker prevent is needed to decrease gastric erosion in this client.
d. H2 blocker and Proton pump inhibitor decreases gastric motility in client with pancreatitis.

10. A client diagnosed with chronic pancreatitis has been taking pancreatic enzymes and H2 blocker for six weeks. Which of the following would indicate that the treatment is effective. Select all that apply?
a. Steady weight gain,
b. Fewer bowel movements
c. Maintain normal blood pressure.
d. Elimination of oil droplet seepage
e. A decrease in stool fat
f. Voiding adequate urine daily.

11. The nurse is performing assessment on a client with tentative diagnosis of acute pancreatitis. Which of the following findings should the nurse anticipate?
   a. Gnawing pain, radiating through to the lower back, with severe abdominal distention.
   b. Pain on light palpation in midepigastric area, chronic low grade fever, and diarrhea.
   c. Increasing abdominal distension, with increased pain and vomiting.
   d. Acute onset of steady, boring upper abdominal pain radiating through to the back.

12. The nurse is planning to insert nasogastric tube for a client with nausea and vomiting.
    The best approach for the nurse to use is (Select all that apply)
    a. Ask the most experienced nurse to perform the procedure.
    b. Explain the procedure to the client
    c. Inspect each nostril for polyps, or lesions.
    d. Place emesis basin within easy reach of the client
    e. Secure the tube to the client’s nose.

13. The home care nurse is visiting a client with chronic pancreatitis who is receiving pancreatic enzyme therapy. Which of the following observations by the nurse would indicate that the chronic pancreatitis is worsening?
    a. Weight gain of 2 pounds in a week
    b. Abdominal pain with eating.
c. Fatty stools of 5 to 8 time per day.
d. Fasting glucose level of 132 mg/dL

14. The nurse is reviewing the care given to a client with acute pancreatitis. The client has a nursing diagnosis of imbalanced nutrition less than body requirement related to increased nutritional needs secondary to acute illness and body temperature. Which of the following responses by the client would indicate to the nurse that the goal has been met?

a. Asks for something to drink
b. Maintains normal serum glucose level
c. Exhibits moist clean oral mucosa
d. Remains alert and responsive

15. The nurse has given discharge teaching to client who is receiving Enzyme replacement therapy for chronic pancreatitis. Which of the following statement by the client would indicate that the client did not understand the nurse’s instructions.

a. “I should take my enzyme replacement therapy pill 30 minutes before I start to eat.”
b. “The enzyme replacement therapy mimic the activity of the pancreas.”
c. “The enzyme replacement therapy delivers active digestive enzyme into the small intestine with food.”
d. “I should take my enzyme replacement therapy with all meals and snacks.”

16. The nurse is planning care for a client with acute pancreatitis. The client has a nursing diagnosis of acute pain and discomfort related to peritoneal irritation and excess stimulation of pancreatic secretion. Which of the following intervention will be essential for the nurse to include in the clients plan of care. Select all that apply.
a. Assess patient pain level before and after administration of analgesics.
b. Maintain the patient on bedrest.
c. Explain rationale for the use of NG tube.
d. Listen to the patient expression of pain experience.
e. Administer anticholinergic medications as ordered.
f. Assess the client’s current nutritional status and increased metabolic requirement.
g. Main patient on nothing by mouth (NPO) as ordered.

17. The nurse is reinforcing diet teaching with a client who has chronic pancreatitis. Which of the following menu choices by the client would indicate a correct understanding of the teaching?

   a. A cheese sandwich, beef bouillon, and sherbet
   b. Creamed chipped beef on toast, tomato juice, and pretzels
   c. Water, chicken salad on whole grain toast, and apples
   d. Apples, Ice cream, a glass of red wine, and garden salad

18. A client with a family history of pancreatic cancer is scheduled to have an ERCP. He is crying as he tells you, “I know that I have pancreatic cancer, too.” Which response is most therapeutic?

   a. “I know just how you feel.”
   b. “You seem upset.”
   c. “Oh, don’t worry about it, everything will be just fine.”
   d. “Why do you think you have cancer?”

19. During the assessment of a client with acute pancreatitis the nurse notes a decrease in breath sounds bilaterally in the lung bases. What should the nurse do with this information?
a. Document the finding.
b. Encourage the client to use the incentive spirometer.
c. Increase the client's intravenous fluids.
d. Report the information to the physician.

20. The nurse is taking care of a patient admitted with acute pancreatitis whose serum glucose level is consistently above 180mg/dL. The nurse will expect the physician to order the following.
   a. Metformin (Glucophage) 500mg orally twice daily.
   b. Beta blocker metoprolol 5 mg orally daily
   c. Regular insulin 10 units SC or IV every morning.
   d. Calcium gluconate 10 mg IV daily
NCLEX Question answers and rationale

1. Rationale: (B, D, A, and C.) Pain prevent the client from taking deep breaths; vomiting leads to fluid and electrolyte imbalance which can worsen the respiration, lead to hypocalcemia tetany, alter neuro function. Administer supplemental oxygen, and complete physical assessment when condition is much stable.

2. Rationale: (B.) Morphine sulfate can be given to the client however oral administration will not be used at this time. Although Morphine sulfate causes spasms of the sphincter of Oddi it is not significant for this client’s pain.

3. Rationale: (D.) Turner sign (ecchymoses of the flanks) and the Cullen sign (ecchymoses of the umbilical region) indicate extravasation of hemorrhagic exudate. Client’s hemoglobin and hematocrit should be evaluated.

4. Rationale: D

5. Rationale: (D.) Local complications in a patient with acute pancreatitis should be suspected when there is persistence or recurrence of abdominal pain, secondary increases in serum pancreatic enzyme activity, increasing organ dysfunction, and/or the development of clinical signs of sepsis, such as fever and leukocytosis. These symptoms if observed should be reported immediately to the physician. Other signs are present in acute pancreatitis.

6. Rationale: (A.) Hypocalcemia generally is not treated unless neuromuscular irritability occurs such as being exhibited by the patient; 10 to 20 mL of 10% Ca gluconate in 1 L of IV fluid is given over 4 to 6 h. the nurse should notify the physician about these symptoms. The nurse has already performed an assessment of the client.

7. Rationale: (A.) If client is unable to speak or has voice hoarseness after insertion of an NG tube. The tube is most likely in the trachea and should be removed and reinserted into the esophagus.

8. Rationale: (C.) during swallowing the epiglottis covers the laryngeal opening so that the NG tube enters the esophagus

9. Rationale: (B.) An H2 blocker or proton pump inhibitor should be given to prevent acid breakdown of the enzymes.
10. Rationale. (A, B, D, E.) Favorable clinical responses to pancreatic enzyme therapy include weight gain, fewer bowel movements, elimination of oil droplet seepage, and improved well-being.

11. Rationale. (D.) The classic sign for acute pancreatitis is abdominal pain (acute onset of a persistent, severe, epigastric pain often radiating to the back).

12. Rationale. All responses are correct.

13. Rationale. (C.) Malabsorption and frequent fatty foul smelling stools occurs late in chronic pancreatitis due to impaired digestion leading to weight loss, this is a sign of worsening pancreatitis. B and c are signs os chronic pancreatitis, A is expected in clients receiving pancreatic enzyme therapy.

14. Rationale. (B.) Impairment of endocrine function of the pancreas leads to increased serum glucose levels, the nurses monitors the serum glucose levels administer prescribed insulin, which drives glucose into the cells for metabolism.

15. Rationale. (A.) Pancreatic enzyme replacement therapies (PERTs) help deliver pancreatic enzyme concentrations into the duodenal lumen to help break down food for absorption. Enzyme replacement therapy should ideally mimic the physiological pattern of pancreatic exocrine secretion, delivering active lipase into the duodenum together with food. Therefore it is important that PERTs are administered together with meals and snacks.

16. Rationale. A, B, D, E, & G. Pain assessment is the foundation for adequate pain management; Anticholinergic medications reduce gastric and pancreatic secretions; bedrest decreases body metabolism; reducing pancreatic and gastric secretion; pancreatic secretion is increased by food and fluid intake. Explaining the rationale for NG tube has no direct effect on the client’s pain.

17. Rationale. (C.) The right diet is important for people with chronic pancreatitis to keep a healthy weight and get the correct nutrients; Drinking plenty of liquids, Limiting fats, Eating small, frequent meals, Getting enough vitamins and calcium in the diet, or as extra supplements, Limiting caffeine, avoiding alcoholic beverages.
18. Rationale. B. Making observations about what you see or hear is a useful therapeutic technique. This way, you acknowledge that you are interested in what the patient is saying and feeling.

19. Rationale. (D.) Atelectasis may result from decreased diaphragmatic excursion because of abdominal distention or from direct injury from exposure to pancreatic enzymes.

20. Rationale. (B.) Endocrine function is affected by pancreatitis. Glucose > 170 to 200 mg/dL (9.4 to 11.1 mmol/L) is treated cautiously with SC or IV insulin and patient carefully monitored.

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