

PANCREATITIS

Pancreatitis is a painful inflammatory condition in which the pancreatic enzymes are prematurely activated resulting in autodigestion of the pancreas. The most common cause of pancreatitis are biliary tract disease and alcoholism, but can also result from such things as abnormal organ structure, blunt trauma, penetrating peptic ulcers, and drugs such as sulfonamides and glucocorticoids. Pancreatitis may be acute or chronic, with symptoms mild to severe.

CARE SETTING

Inpatient acute medical unit for initial incident or exacerbations with serious complications; otherwise condition is managed at the community level.

RELATED CONCERNS

Alcoholism (acute); intoxication/overdose
Substance dependence/abuse rehabilitation
Diabetes mellitus/diabetic ketoacidosis
Peritonitis
Psychosocial aspects of care
Renal failure: acute
Sepsis/septicemia
Total nutritional support; parenteral/enteral feeding

Patient Assessment Database

CIRCULATION

May exhibit: Hypertension (acute pain); hypotension and tachycardia (hypovolemic shock or toxemia)
Edema, ascites
Skin pale, cold, mottled with diaphoresis (vasoconstriction/fluid shifts); jaundiced (inflammation/ obstruction of common duct); blue-green-brown discoloration around umbilicus (Cullen's sign) from accumulation of blood (hemorrhagic pancreatitis)

EGO INTEGRITY

May exhibit: Agitation, restlessness, distress, apprehension

ELIMINATION

May report: Diarrhea
May exhibit: Bowel sounds decreased/absent (reduced peristalsis/ileus)
Dark amber or brown, foamy urine (bile)
Frothy, foul-smelling, grayish, greasy, nonformed stool (steatorrhea)
Polyuria (developing DM)

FOOD/FLUID

May report: Food intolerance, anorexia; frequent/persistent vomiting, retching, dry heaves
Weight loss
May exhibit: Diffuse epigastric/abdominal tenderness to palpation, abdominal rigidity, distension
Hypoactive bowel sounds
Urine positive for glucose

NEUROSENSORY

May exhibit: Confusion, agitation
Coarse tremors of extremities (hypocalcemia)

PAIN/DISCOMFORT

- May report:** Unrelenting severe deep abdominal pain, usually located in the epigastrium and periumbilical regions but may radiate to the back; onset may be sudden and often associated with heavy drinking or a large meal
Radiation to chest and back, may increase in supine position
- May exhibit:** Abdominal guarding, may curl up on left side with both arms over abdomen and knees/hips flexed
Abdominal rigidity

RESPIRATION

- May exhibit:** Tachypnea, with/without dyspnea
Decreased depth of respiration with splinting/guarding actions
Bibasilar crackles (pleural effusion)

SAFETY

- May exhibit:** Fever

SEXUALITY

- May exhibit:** Current pregnancy (third trimester) with shifting of abdominal contents and compression of biliary tract

TEACHING/LEARNING

- May report:** Family history of pancreatitis
Diabetic ketoacidosis
History of cholelithiasis with partial or complete common bile duct obstruction; gastritis, duodenal ulcer, duodenitis; diverticulitis; Crohn's disease; recent abdominal surgery (e.g., procedures on the pancreas, biliary tract, stomach, or duodenum); external abdominal trauma
Excessive alcohol intake (90% of cases)
Uses of medications, e.g., salicylates, pentamidine, antihypertensives, opiates, thiazides, steroids, some antibiotics, estrogens
Infectious diseases, e.g., mumps, hepatitis B, Coxsackie viral infection
- Discharge plan considerations:** **DRG projected mean length of inpatient stay: 5.7 days**
May require assistance with dietary program, homemaker/maintenance tasks
Refer to section at end of plan for postdischarge considerations

DIAGNOSTIC STUDIES

- CT scan:** Shows an enlarged pancreas, pancreatic cysts and determines extent of edema and necrosis.
- Ultrasound of abdomen:** May be used to identify pancreatic inflammation, abscess, pseudocysts, carcinoma, or obstruction of biliary tract
- Endoscopic retrograde cholangiopancreatography:** Useful to diagnose fistulas, obstructive biliary disease, and pancreatic duct strictures/anomalies (procedure is contraindicated in acute phase).
- CT-guided needle aspiration:** Done to determine whether infection is present.
- Abdominal x-rays:** May demonstrate dilated loop of small bowel adjacent to pancreas or other intra-abdominal precipitator of pancreatitis, presence of free intraperitoneal air caused by perforation or abscess formation, pancreatic calcification.
- Upper GI series:** Frequently exhibits evidence of pancreatic enlargement/inflammation.
- Serum amylase:** Increased because of obstruction of normal outflow of pancreatic enzymes (normal level does not rule out disease). May be five or more times normal level in acute pancreatitis.
- Serum lipase:** usually elevates along with amylase, but stays elevated longer.
- Serum bilirubin:** Elevation is common (may be caused by alcoholic liver disease or compression of common bile duct).
- Alkaline phosphatase:** Usually elevated if pancreatitis is accompanied by biliary disease.
- Serum albumin and protein:** May be decreased (increased capillary permeability and transudation of fluid into extracellular space).
- Serum calcium:** Hypocalcemia may appear 2–3 days after onset of illness (usually indicates fat necrosis and may accompany pancreatic necrosis).
- Potassium:** Hypokalemia may occur because of gastric losses; hyperkalemia may develop secondary to tissue necrosis, acidosis, renal insufficiency.

Triglycerides: Levels may exceed 1700 mg/dL and may be causative agent in acute pancreatitis.

LDH/AST: May be elevated up to 15 times normal because of biliary and liver involvement.

CBC: WBC count of 10,000–25,000 is present in 80% of patients. Hb may be lowered because of bleeding. Hct is usually elevated (hemoconcentration associated with vomiting or from effusion of fluid into pancreas or retroperitoneal area).

Serum glucose: Transient elevations of more than 200 mg/dL are common, especially during initial/acute attacks.

Sustained hyperglycemia reflects widespread cell damage and pancreatic necrosis and is a poor prognostic sign.

Partial thromboplastin time (PTT): Prolonged if coagulopathy develops because of liver involvement and fat necrosis.

Urinalysis: Glucose, myoglobin, blood, and protein may be present.

Urine amylase: Can increase dramatically within 2–3 days after onset of attack.

Stool: Increased fat content (steatorrhea) indicative of insufficient digestion of fats and protein.

NURSING PRIORITIES

1. Control pain and promote comfort.
2. Prevent/threat fluid and electrolyte imbalance.
3. Reduce pancreatic stimulation while maintaining adequate nutrition.
4. Prevent complications.
5. Provide information about disease process/prognosis and treatment needs.

DISCHARGE GOALS

1. Pain relieved/controlled.
2. Hemodynamically stable.
3. Complications prevented/minimized.
4. Disease process/prognosis, potential complications, and therapeutic regimen understood.
5. Plan in place to meet needs after discharge.

NURSING DIAGNOSIS: Pain, acute

May be related to

Obstruction of pancreatic, biliary ducts

Chemical contamination of peritoneal surfaces by pancreatic exudate/autodigestion of pancreas

Extension of inflammation to the retroperitoneal nerve plexus

Possibly evidenced by

Reports of pain

Self-focusing, grimacing, distraction/guarding behaviors

Autonomic responses, alteration in muscle tone

DESIRED OUTCOMES/EVALUATION CRITERIA—PATIENT WILL:

Pain Control (NOC)

Report pain is relieved/controlled.

Follow prescribed therapeutic regimen.

Demonstrate use of methods that provide relief.

ACTIONS/INTERVENTIONS	RATIONALE
<p>Pain Management (NIC)</p> <p>Independent</p> <p>Investigate verbal reports of pain, noting specific location and intensity (0–10 scale). Note factors that aggravate and relieve pain.</p> <p>Maintain bedrest during acute attack. Provide quiet, restful environment.</p> <p>Promote position of comfort, e.g., on one side with knees flexed, sitting up and leaning forward.</p> <p>Provide alternative comfort measures (e.g., back rub), encourage relaxation techniques (e.g., guided imagery, visualization), quiet diversional activities (e.g., TV, radio).</p> <p>Keep environment free of food odors.</p> <p>Administer analgesics in timely manner (smaller, more frequent doses).</p> <p>Maintain meticulous skin care, especially in presence of draining abdominal wall fistulas.</p>	<p>Pain is often diffuse, severe, and unrelenting in acute or hemorrhagic pancreatitis. Severe pain is often the major symptom in patients with chronic pancreatitis. Isolated pain in the RUQ reflects involvement of the head of the pancreas. Pain in the left upper quadrant (LUQ) suggests involvement of the pancreatic tail. Localized pain may indicate development of pseudocysts or abscesses.</p> <p>Decreases metabolic rate and GI stimulation/secretions, thereby reducing pancreatic activity.</p> <p>Reduces abdominal pressure/tension, providing some measure of comfort and pain relief. <i>Note:</i> Supine position often increases pain.</p> <p>Promotes relaxation and enables patient to refocus attention; may enhance coping.</p> <p>Sensory stimulation can activate pancreatic enzymes, increasing pain.</p> <p>Severe/prolonged pain can aggravate shock and is more difficult to relieve, requiring larger doses of medication, which can mask underlying problems/complications and may contribute to respiratory depression.</p> <p>Pancreatic enzymes can digest the skin and tissues of the abdominal wall, creating a chemical burn.</p>
<p>Collaborative</p> <p>Administer medication as indicated:</p> <p>Narcotic analgesics, e.g., meperidine (Demerol), fentanyl (Sublimaze), pentazocine (Talwin);</p> <p>Sedatives, e.g., diazepam (Valium); antispasmodics, e.g., atropine;</p> <p>Antacids, e.g., Mylanta, Maalox, Amphojel, Riopan;</p>	<p>Meperidine is usually effective in relieving pain and may be preferred over morphine, which can have a side effect of biliary-pancreatic spasms. Paravertebral block has been used to achieve prolonged pain control. <i>Note:</i> Pain in patients who have recurrent or chronic pancreatitis episodes may be difficult to manage because they may become dependent on the narcotics given for pain control.</p> <p>Potentiates action of narcotic to promote rest and to reduce muscular/ductal spasm, thereby reducing metabolic needs, enzyme secretions.</p> <p>Neutralizes gastric acid to reduce production of pancreatic enzymes and to reduce incidence of upper GI bleeding.</p>

ACTIONS/INTERVENTIONS	RATIONALE
<p>Pain Management (NIC)</p> <p>Independent</p> <p>Cimetidine (Tagamet), ranitidine (Zantac), famotidine (Pepcid).</p> <p>Withhold food and fluid as indicated.</p> <p>Maintain gastric suction when used.</p> <p>Prepare for surgical intervention if indicated.</p>	<p>Decreasing secretion of HCl reduces stimulation of the pancreas and associated pain.</p> <p>Limits/reduces release of pancreatic enzymes and resultant pain.</p> <p>Prevents accumulation of gastric secretions, which can stimulate pancreatic enzyme activity.</p> <p>Surgical exploration may be required in presence of intractable pain/complications involving the biliary tract, such as pancreatic abscess or pseudocyst.</p>

<p>NURSING DIAGNOSIS: Fluid Volume, risk for deficient</p> <p>Risk factors may include</p> <p>Excessive losses: vomiting, gastric suctioning</p> <p>Increase in size of vascular bed (vasodilation, effects of kinins)</p> <p>Third-space fluid transudation, ascites formation</p> <p>Alteration of clotting process, hemorrhage</p> <p>Possibly evidenced by</p> <p>[Not applicable; presence of signs and symptoms establishes an <i>actual</i> diagnosis.]</p> <p>DESIRED OUTCOMES/EVALUATION CRITERIA—PATIENT WILL:</p> <p>Hydration (NOC)</p> <p>Maintain adequate hydration as evidenced by stable vital signs, good skin turgor, prompt capillary refill, strong peripheral pulses, and individually appropriate urinary output.</p>

ACTIONS/INTERVENTIONS	RATIONALE
<p>Fluid/Electrolyte Management (NIC)</p> <p>Independent</p> <p>Monitor BP and measure CVP if available.</p> <p>Measure I&O including vomiting/gastric aspirate, diarrhea. Calculate 24-hr fluid balance.</p>	<p>Fluid sequestration (shifts into third space), bleeding, and release of vasodilators (kinins) and cardiac depressant factor triggered by pancreatic ischemia may result in profound hypotension. Reduced cardiac output/poor organ perfusion secondary to a hypotensive episode can precipitate widespread systemic complications.</p> <p>Indicators of replacement needs/effectiveness of therapy.</p>

ACTIONS/INTERVENTIONS	RATIONALE
<p>Fluid/Electrolyte Management (NIC)</p> <p>Independent</p> <p>Note decrease in urine output (less than 400 mL/24 hr).</p> <p>Record color and character of gastric drainage, measure pH, and note presence of occult blood.</p> <p>Weigh as indicated. Correlate with calculated fluid balance.</p> <p>Note poor skin turgor, dry skin/mucous membranes, reports of thirst.</p> <p>Observe/record peripheral and dependent edema. Measure abdominal girth if ascites present.</p> <p>Investigate changes in sensorium, e.g., confusion, slowed responses.</p> <p>Auscultate heart sounds; note rate and rhythm. Monitor/document rhythm, changes.</p> <p>Inspect skin for petechiae, hematomas, and unusual wound or venipuncture bleeding. Note hematuria, mucous membrane bleeding, and bloody gastric contents.</p> <p>Observe/report coarse muscle tremors, twitching, positive Chvostek's or Trousseau's sign.</p>	<p>Oliguria may occur, signaling renal impairment/acute tubular necrosis (ATN), related to increase in renal vascular resistance or reduced/altered renal blood flow.</p> <p>Risk of gastric bleeding/hemorrhage is high.</p> <p>Weight loss may suggest hypovolemia; however, edema, fluid retention, and ascites may be reflected by increased or stable weight, even in the presence of muscle wasting.</p> <p>Further physiological indicators of dehydration.</p> <p>Edema/fluid shifts occur as a result of increased vascular permeability, sodium retention, and decreased colloid osmotic pressure in the intravascular compartment. <i>Note:</i> Fluid loss (sequestration) of more than 6 L/48 hr is considered a poor prognostic sign.</p> <p>Changes may be related to hypovolemia, hypoxia, electrolyte imbalance, or impending delirium tremens (in patient with acute pancreatitis secondary to excessive alcohol intake). Severe pancreatic disease may cause toxic psychosis.</p> <p>Cardiac changes/dysrhythmias may reflect hypovolemia and/or electrolyte imbalance, commonly hypokalemia/hypocalcemia. Hyperkalemia may occur related to tissue necrosis, acidosis, and renal insufficiency and may precipitate lethal dysrhythmias if uncorrected. S₃ gallop in conjunction with JVD and crackles suggest HF/pulmonary edema. <i>Note:</i> Cardiovascular complications are common and include MI, pericarditis, and pericardial effusion with/without tamponade.</p> <p>DIC may be initiated by release of active pancreatic proteases into the circulation. The most frequently affected organs are the kidneys, skin, and lungs.</p> <p>Symptoms of calcium imbalance. Calcium binds with free fats in the intestine and is lost by excretion in the stool.</p>

ACTIONS/INTERVENTIONS	RATIONALE
<p>Fluid/Electrolyte Management (NIC)</p> <p>Collaborative</p> <p>Administer fluid replacement as indicated, e.g., saline solutions, albumin, blood/blood products, dextran.</p> <p>Monitor laboratory studies, e.g., Hb/Hct, Protein, albumin, electrolytes, BUN, creatinine, urine osmolality and sodium/potassium, coagulation studies.</p> <p>Replace electrolytes, e.g., sodium, potassium, chloride, calcium as indicated.</p> <p>Prepare for/assist with peritoneal lavage, hemoperitoneal dialysis.</p>	<p>Choice of replacement solution may be less important than rapidity and adequacy of volume restoration. Saline solutions and albumin may be used to promote mobilization of fluid back into vascular space. Low-molecular-weight dextran is sometimes used to reduce risk of renal dysfunction and pulmonary edema associated with pancreatitis.</p> <p>Identifies deficits/replacement needs and developing complications, e.g., ATN, DIC.</p> <p>Decreased oral intake and excessive losses greatly affect electrolyte/acid-base balance, which is necessary to maintain optimal cellular/organ function.</p> <p>Removes toxic chemicals/pancreatic enzymes and allows for more rapid correction of metabolic abnormalities in severe/unresponsive cases of acute pancreatitis.</p>

<p>NURSING DIAGNOSIS: Nutrition: imbalanced, less than body requirements</p> <p>May be related to</p> <p>Vomiting, decreased oral intake; prescribed dietary restrictions</p> <p>Loss of digestive enzymes and insulin (related to pancreatic outflow obstruction or necrosis/autodigestion)</p> <p>Possibly evidenced by</p> <p>Reported inadequate food intake</p> <p>Aversion to eating, reported altered taste sensation, lack of interest in food</p> <p>Weight loss</p> <p>Poor muscle tone</p> <p>DESIRED OUTCOMES/EVALUATION CRITERIA—PATIENT WILL:</p> <p>Nutritional Status (NOC)</p> <p>Demonstrate progressive weight gain toward goal with normalization of laboratory values</p> <p>Experience no signs of malnutrition.</p> <p>Knowledge: Diet (NOC)</p> <p>Demonstrate behaviors, lifestyle changes to regain and/or maintain appropriate weight.</p>

ACTIONS/INTERVENTIONS	RATIONALE
<p>Nutrition Management (NIC)</p> <p>Independent</p> <p>Assess abdomen, noting presence/character of bowel sounds, abdominal distension, and reports of nausea.</p> <p>Provide frequent oral care.</p> <p>Assist patient in selecting food/fluids that meet nutritional needs and restrictions when diet is resumed.</p> <p>Observe color/consistency/amount of stools. Note frothy consistency/foul odor.</p>	<p>Gastric distention and intestinal atony are frequently present, resulting in reduced/absent bowel sounds. Return of bowel sounds and relief of symptoms signal readiness for discontinuation of gastric aspiration (NG tube).</p> <p>Decreases vomiting stimulus and inflammation/irritation of dry mucous membranes associated with dehydration and mouth breathing when NG is in place.</p> <p>Previous dietary habits may be unsatisfactory in meeting current needs for tissue regeneration and healing. Use of gastric stimulants, e.g., caffeine, alcohol, cigarettes, gas-producing foods, or ingestion of large meals may result in excessive stimulation of the pancreas/recurrence of symptoms.</p> <p>Steatorrhea may develop from incomplete digestion of fats.</p>
<p>Hyperglycemia Management (NIC)</p> <p>Note signs of increased thirst and urination or changes in mentation and visual acuity.</p> <p>Test urine for sugar and acetone.</p> <p>Maintain NPO status and gastric suctioning in acute phase.</p> <p>Administer hyperalimentation and lipids, if indicated.</p> <p>Resume oral intake with clear liquids and advance diet slowly to provide high-protein, high-carbohydrate diet, when indicated.</p>	<p>May warn of developing hyperglycemia associated with increased release of glucagon (damage to [beta] cells) or decreased release of insulin (damage to [beta] cells).</p> <p>Early detection of inadequate glucose utilization may prevent development of ketoacidosis.</p> <p>Prevents stimulation and release of pancreatic enzymes (secretin), released when chyme and HCl enter the duodenum.</p> <p>IV administration of calories, lipids, and amino acids should be instituted before nutrition/nitrogen depletion is advanced.</p> <p>Oral feedings given too early in the course of illness may exacerbate symptoms. Loss of pancreatic function/reduced insulin production may require initiation of a diabetic diet.</p>
<p>Nutrition Management (NIC)</p> <p>Collaborative</p> <p>Provide medium-chain triglycerides (MCTs) (e.g., MCT, Portagen).</p>	<p>MCTs are elements of enteral feedings (NG or J-tube) that provide supplemental calories/nutrients that do not require pancreatic enzymes for digestion/absorption.</p>

ACTIONS/INTERVENTIONS	RATIONALE
<p>Nutrition Management (NIC)</p> <p>Collaborative</p> <p>Administer medications as indicated: Vitamins, e.g., A,D,E,K;</p> <p>Replacement enzymes, e.g., pancreatin (Dizymes), pancrelipase (Viokase, Cotazym).</p> <p>Hyperglycemia Management (NIC)</p> <p>Monitor serum glucose.</p> <p>Provide insulin as appropriate.</p>	<p>Replacement required because fat metabolism is altered, reducing absorption/storage of fat-soluble vitamins.</p> <p>Used in chronic pancreatitis to correct deficiencies to promote digestion and absorption of nutrients.</p> <p>Indicator of insulin needs because hyperglycemia is frequently present, although not usually in levels high enough to produce ketoacidosis.</p> <p>Corrects persistent hyperglycemia caused by injury to cells and increased release of glucocorticoids. Insulin therapy is usually short-term unless permanent damage to pancreas occurs.</p>

<p>NURSING DIAGNOSIS: Infection, risk for</p> <p>Risk factors may include</p> <p>Inadequate primary defenses: stasis of body fluids, altered peristalsis, change in pH of secretions</p> <p>Immunosuppression</p> <p>Nutritional deficiencies</p> <p>Tissue destruction, chronic disease</p> <p>Possibly evidenced by:</p> <p>[Not applicable; presence of signs and symptoms establishes an <i>actual</i> diagnosis.]</p> <p>DESIRED OUTCOMES/EVALUATION CRITERIA—PATIENT WILL:</p> <p>Immune Status (NOC)</p> <p>Achieve timely healing, be free of signs of infection.</p> <p>Be afebrile</p> <p>Risk Control (NOC)</p> <p>Participate in activities to reduce risk of infection.</p>
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ACTIONS/INTERVENTIONS	RATIONALE
<p>Infection Protection (NIC)</p> <p>Independent</p> <p>Use strict aseptic technique when changing surgical dressings or working with IV lines, indwelling catheters/tubes, drains. Change soiled dressings promptly.</p> <p>Stress importance of good handwashing.</p> <p>Observe rate and characteristics of respirations, breath sounds. Note occurrence of cough and sputum production.</p> <p>Encourage frequent position changes, deep breathing, and coughing. Assist with ambulation as soon as stable.</p> <p>Observe for signs of infection, e.g.: Fever and respiratory distress in conjunction with jaundice;</p> <p>Increased abdominal pain, rigidity/rebound tenderness, diminished/absent bowel sounds;</p> <p>Increased abdominal pain/tenderness, recurrent fever (higher than 101°F), leukocytosis, hypotension, tachycardia, and chills.</p>	<p>Limits sources of infection, which can lead to sepsis in a compromised patient. <i>Note:</i> Studies indicate that infectious complications are responsible for about 80% of deaths associated with pancreatitis.</p> <p>Reduces risk of cross-contamination.</p> <p>Fluid accumulation and limited mobility predispose to respiratory infections and atelectasis. Accumulation of ascites fluid may cause elevated diaphragm and shallow abdominal breathing.</p> <p>Enhances ventilation of all lung segments and promotes mobilization of secretions.</p> <p>Cholestatic jaundice and decreased pulmonary function may be first sign of sepsis involving Gram-negative organisms.</p> <p>Suggestive of peritonitis.</p> <p>Abscesses can occur 2 wk or more after the onset of pancreatitis (mortality can exceed 50%) and should be suspected whenever patient is deteriorating despite supportive measures.</p>
<p>Collaborative</p> <p>Obtain culture specimens, e.g., blood, wound, urine, sputum, or pancreatic aspirate.</p> <p>Administer antibiotic therapy as indicated, e.g.,: cephalosporins, cefoxitin sodium (Mefoxin); plus aminoglycosides, e.g., gentamicin (Garamycin), tobramycin (Nebcin).</p> <p>Prepare for surgical intervention as necessary.</p>	<p>Identifies presence of infection and causative organism.</p> <p>Broad-spectrum antibiotics are generally recommended for sepsis; however, therapy will be based on the specific organisms cultured.</p> <p>Abscesses may be surgically drained with resection of necrotic tissue. Sump tubes may be inserted for antibiotic irrigation and drainage of pancreatic debris. Pseudocysts (persisting for several weeks), may be drained because of the risk and incidence of infection/rupture.</p>

NURSING DIAGNOSIS: Knowledge, deficient [Learning Need] regarding condition, prognosis, treatment, self-care, and discharge needs

May be related to

Lack of exposure/recall
Information misinterpretation; unfamiliarity with information resources

Possibly evidenced by

Questions, request for information; statement of misconception
Inaccurate follow-through of instructions/development of preventable complication

DESIRED OUTCOMES/EVALUATION CRITERIA—PATIENT WILL:

Knowledge: Illness Care (NOC)

Verbalize understanding of condition/disease process and potential complications.
Verbalize understanding of therapeutic needs.
Correctly perform necessary procedures and explain reasons for the actions.
Initiate necessary lifestyle changes and participate in treatment regimen.

ACTIONS/INTERVENTIONS	RATIONALE
<p>Teaching: Disease Process (NIC)</p>	
<p>Independent</p>	
<p>Review specific cause of current episode and prognosis.</p>	<p>Provides knowledge base on which patient can make informed choices.</p>
<p>Discuss other causative/associated factors, e.g., excessive alcohol intake, gallbladder disease, duodenal ulcer, hyperlipoproteinemias, some drugs (e.g., oral contraceptives, thiazides, furosemide [Lasix], isoniazid [INH], glucocorticoids, sulfonamides).</p>	<p>Avoidance may help limit damage and prevent development of a chronic condition.</p>
<p>Explore availability of treatment programs/rehabilitation of chemical dependency if indicated.</p>	<p>Alcohol abuse is currently the most common cause of recurrence of chronic pancreatitis. Usage of other drugs, whether prescribed or illicit, is increasing as a factor. <i>Note:</i> Pain of pancreatitis can be severe and prolonged and may lead to narcotic dependence, requiring need for referral to pain clinic.</p>
<p>Stress the importance of follow-up care, and review symptoms that need to be reported immediately to physician, e.g., recurrence of pain, persistent fever, nausea/vomiting, abdominal distension, frothy/foul-smelling stools, general intolerance of food.</p>	<p>Prolonged recovery period requires close monitoring to prevent recurrence/complications, e.g., infection, pancreatic pseudocysts.</p>
<p>Review importance of initially continuing bland, low-fat diet with frequent small feedings and restricted caffeine, with gradual resumption of a normal diet within individual tolerance.</p>	<p>Understanding the purpose of the diet in maximizing the use of available enzymes while avoiding overstimulation of the pancreas may enhance patient involvement in self-monitoring of dietary needs and responses to foods.</p>

ACTIONS/INTERVENTIONS	RATIONALE
<p>Teaching: Disease Process (NIC)</p> <p>Independent</p> <p>Instruct in use of pancreatic enzyme replacements and bile salt therapy as indicated, avoiding concomitant ingestion of hot foods/fluids.</p> <p>Recommend cessation of smoking.</p> <p>Discuss signs/symptoms of diabetes mellitus, i.e., polydipsia, polyuria, weakness, weight loss.</p>	<p>If permanent damage to the pancreas has occurred, exocrine deficiencies will occur, requiring long-term replacement. Hot foods/fluids can inactivate enzymes.</p> <p>Nicotine stimulates gastric secretions and unnecessary pancreatic activity.</p> <p>Damage to the [beta] cells may result in a temporary or permanent alteration of insulin production.</p>

POTENTIAL CONSIDERATIONS following acute hospitalization (dependent on patient's age, physical condition/presence of complications, personal resources, and life responsibilities)

Nutrition: imbalanced, less than body requirements—preexisting malnutrition, prescribed dietary restrictions, persistent nausea/vomiting, imbalances in digestive enzymes.

Pain, acute/chronic—chemical irritation of peritoneal surfaces by pancreatic enzymes, spasms of biliary ducts, general inflammatory process.

Family Processes, dysfunctional: alcoholism—abuse of alcohol, resistance to treatment, inadequate coping/lack of problem-solving skills, addictive personality/codependency.

Therapeutic Regimen: ineffective management—complexity of therapeutic regimen, economic difficulties, mistrust of regimen, perceived benefit, social support deficits.