Chronic Pancreatitis — Learn About the Pathophysiology, Symptoms, Causes, and Treatment Options, as Well as MNT to Optimize Nutrition Status in Patients
By Megan Baumler, PhD, RD

Suggested CDR Learning Codes: 3005, 3020, 5000, 5220; Level 3

Suggested CDR Performance Indicators: 8.1.5, 10.2.5, 10.2.8, 10.2.9

A 45-year-old male is admitted to the emergency department complaining of severe abdominal pain. This is his fourth emergency department admission in eight months for abdominal pain. The previous admissions resulted in a pancreatitis diagnosis, which spontaneously resolved. This visit again results in a pancreatitis diagnosis, and he is admitted to the hospital. Following a CT scan, the attending physician explains to the patient that he sees morphological changes in the pancreas that indicate fibrosis. The physician diagnoses the man with chronic pancreatitis and orders a nutrition consult.

Pancreatitis is the most common disease that affects the pancreas and is characterized by inflammation of the organ. Pancreatitis may be acute, most cases of which resolve without consequence, although a small percentage of acute pancreatitis cases result in death. Chronic pancreatitis, in comparison, is ongoing inflammation resulting in repeated injury to the tissue and morphologic changes that irreversibly interfere with the normal function of the pancreas.¹

Incidence of chronic pancreatitis is estimated to be four out of every 100,000 people in the United States.² In 2009, chronic pancreatitis caused 19,724 hospitalizations and resulted in health care costs of $172 million.³ Chronic pancreatitis has a critical impact on nutritional status because of the central role the pancreas plays in digestion and glucose homeostasis.

The pancreas is an organ with both endocrine (secretion into bloodstream) and exocrine (secretion into ducts) functions. Although the islets of Langerhans, the portion of the gland that contains alpha and beta cells that secrete hormones into the bloodstream, make up only 1% to 2% of the organ, the endocrine responsibilities of the pancreas may be better known because of their relationship to diabetes.⁴ Alpha and beta cells regulate serum insulin by secreting glucagon and insulin, respectively. The other 98% of pancreatic tissue contains cells that are involved in synthesis and secretion of digestive enzymes to facilitate digestion and food absorption. The pancreas has the highest rate of protein synthesis of any other organ or tissue in the body.⁵ The digestive enzymes are synthesized in acinar cells in an inactive form and secreted into the bile duct upon consumption of a meal. From the bile duct, the enzymes are emptied into the proximal duodenum and quickly activated by an enzyme present on the microvilli of the cells lining the small intestine, known as enteropeptidase, to facilitate digestion of the meal so that the carbohydrates, amino acids, and lipids can be absorbed. If the enzymes are aberrantly activated within the organ, the enzymes digest the pancreatic tissue, called autodigestion, and cause inflammation.⁶
Normally, the pancreas is protected against autodigestion, but certain situations such as bile duct obstruction, alcoholism, congenital abnormalities, and specific genetic mutations increase risk of early activation of the zymogens (inactive digestive enzymes) before their secretion into the bile duct. Pancreatic inflammation, or pancreatitis, interferes with the exocrine, and, over time, endocrine, roles of the pancreas, ultimately leading to malnutrition due to insufficient digestive enzymes, and diabetes. Unfortunately, there’s no intervention or cure for pancreatitis other than pharmacological treatment for pain control and management of subsequent complications such as enzyme insufficiency, malnutrition, and diabetes. Furthermore, patients with chronic pancreatitis usually require pancreatic enzyme replacement therapy (PERT) for digestive enzyme insufficiency and intervention for diabetes. Due to the major role that the pancreas plays in digestion and the significant nutritional implications of pancreatitis, dietitians must be familiar with the pathophysiology of the disease and the current recommendations for MNT.

**Inherent Protection Against Pancreatitis**

The pancreas, which is beneath the stomach and next to the duodenum, is made of different types of cells that are homogenously distributed among the head and tail of the organ. The alpha and beta islet cells carry out the endocrine function by synthesizing the metabolic hormones glucagon and insulin, respectively, which are released into the blood stream to maintain serum glucose homeostasis. Acinar cells are clustered like grapes around ductules, which eventually join to form the bile duct. Acinar cells synthesize and secrete inactive digestive enzymes such as lipase, amylase, and trypsinogen, along with water and bicarbonate.

When dietary carbohydrates, lipids, and proteins enter the small intestine, neural and endocrine cells lining the duodenum are stimulated to secrete cholecystokinin and acetylcholine into the bloodstream, which stimulate the acinar cells of the pancreas. Upon stimulation, acinar cells secrete the inactive digestive enzymes into the ductules, which eventually empty into the duodenum via the bile duct. Once in the duodenum, the inactive trypsinogen is activated by enteropeptidase, an enzyme in the brush border of the duodenum. Trypsin, an active proteolytic (protein-digesting) enzyme, activates the other enzymes, including lipase, amylase, and carboxypeptidase. Once activated, the enzymes hydrolyze dietary carbohydrates, peptides, and lipids into glucose, amino acids, and fatty acids, respectively, that are absorbed into the single layer of epithelial cells that separate the lumen of the gastrointestinal tract from the bloodstream. These nutrients migrate through the cells and enter the bloodstream, from which the body may use them as needed.

Under normal physiologic circumstances, the pancreas is protected against autodigestion, or self-digestion of the organ, by active digestive enzymes through three mechanisms. First, the enzymes are synthesized in an inactive form, so they cannot digest the organ itself. Second, the inactive enzymes, or zymogens, are contained in a separate cellular compartment within the acinar cells, called zymogen granules. This prevents the exposure of the zymogens to hydrolases or other housecleaning enzymes that could activate the zymogens. Finally, a protective molecule called the trypsin inhibitor, which is present within acinar cells, inactivates small amounts of active trypsin before it causes any damage. Normally, a small amount of trypsinogen is activated into trypsin within acinar cells. The trypsin inhibitor is able to prevent
autodigestion as long as the active trypsin is at its normal low levels. Under conditions of pancreatitis, the larger amount of aberrantly activated trypsin is too much for the trypsin inhibitor.4

Pathophysiology and Etiology
Pancreatitis is caused by the premature activation of trypsin within the pancreas itself, leading to autodigestion. Pancreatic autodigestion results in severe abdominal pain, and, over time, chronic inflammation that causes irreversible morphological changes and fibrosis.4 The inflammatory response causes the replacement of acinar and islet cells with nonfunctional fibrotic cells, leading to reduced pancreatic function. Aside from fibrosis, other morphological changes include calcifications that also may develop within the organ.4 Fortunately, the pancreas is an organ of great reserve, meaning that almost 90% of function can be lost before there’s any clinical manifestation.1

While the etiology of pancreatitis affects how quickly the disease progresses, those with chronic pancreatitis caused by alcoholism develop exocrine insufficiency, on average, approximately five years after diagnosis.11 The progressive nature of chronic pancreatitis eventually results in the inability to produce enough digestive enzymes to adequately digest the amount of food usually consumed. The endocrine pancreas also is affected and the majority of people with chronic pancreatitis develop pancreatogenic diabetes due to loss of islet cells.12 Diabetes is classified as pancreatogenic if it’s caused by pancreatic disease, as opposed to an autoimmune disease as in the case of type 1, or metabolic derangement, as in the case of type 2.12 The majority of pancreatogenic diabetes cases are caused by chronic pancreatitis.12

In chronic pancreatitis, trypsin may be aberrantly and continuously activated within the organ by a number of offenders that fall into four categories: toxic metabolites, genetic mutations, autoimmune disease, and long-term obstruction. There are also a significant number of unexplained cases of chronic pancreatitis for which the etiology is classified as idiopathic.

The most common cause of continuous, chronic inflammation of the pancreas in the United States is alcoholism.13 Alcoholic chronic pancreatitis is more common in males, and the typical age at which diagnosis is made is around 40 to 50 years old.4 The exact mechanisms behind the relationship between alcoholism and pancreatitis are unclear, but chronic exposure of the pancreas to the toxic metabolic byproducts of alcohol metabolism has been shown to increase oxidative stress, leading to necrosis (tissue death), and cytokine production.13 Cytokines cause activation of stellate cells and the formation of fibrotic tissue which, over time, replaces the acinar and islet cells.14 One study estimated that 80 g of alcohol, or roughly eight standard alcoholic beverages per day for six to 12 years, is needed to cause pancreatitis.15 While the majority of people with chronic pancreatitis struggle with alcoholism, less than 10% of people with alcoholism develop pancreatitis, indicating that there’s an additional factor or factors working in conjunction with alcoholism that must be present to cause the disease.12,15 The inflammation caused by alcohol exposure may sensitize the pancreas to other triggers such as poor diet, nicotine intake, and genetic predisposition. Nonetheless, complete abstinence from alcohol is paramount to delay progression of alcoholic pancreatitis.16
Other etiological factors in the toxic metabolites category include cigarette smoking and diet. In decades past, there was a known association between cigarette smoking and risk of chronic pancreatitis, but not until recently was this association shown to be causal. The North American Pancreatitis Study, a multicenter study in the United States that included 540 patients with chronic pancreatitis, identified smoking as an independent risk factor for pancreatitis, since those who smoked longer and more heavily were at greater risk of pancreatitis when all other variables were equal.\(^{17}\) In another study, chronic pancreatitis patients who smoked had more pancreatic calcifications and diabetes diagnoses than did nonsmokers, regardless of alcohol intake.\(^{18}\) One study, after controlling for alcohol intake, demonstrated that people who smoked had double the risk of chronic pancreatitis than nonsmokers.\(^{19}\)

Dietary fat and protein are the macronutrients that stimulate pancreatic secretion, but whether diet itself is an independent risk factor for chronic pancreatitis is unclear. In one of the few studies that examined diet as a possible risk factor for chronic pancreatitis, researchers conducted a cross-sectional case study to examine the relationship between fat intake and age of chronic pancreatitis diagnosis and development of complications.\(^{20}\) Of the 168 patients in the study, 24 followed a high-fat diet, had a significantly younger average age of diagnosis (37 years compared with 46 years), and were more likely to have continuous abdominal pain compared with those on a lower fat diet. More studies on diet as an independent risk factor for chronic pancreatitis are needed before researchers can draw any conclusions.

In the last couple of decades, several hereditary factors have been recognized to predispose risk of chronic pancreatitis. For individuals with genetic mutations that cause pancreatitis, onset of the disease usually occurs early, before the age of 10, as opposed to the later onset typical with alcoholism-related chronic pancreatitis.\(^{21}\) The majority of hereditary chronic pancreatitis patients have a mutation in the trypsinogen gene.\(^{22}\) Several mutations in this gene, which codes for the digestive enzyme trypsin, are associated with risk of chronic pancreatitis. The most common mutation within this gene that causes chronic pancreatitis affects a site on the enzyme where it’s cleaved to become inactivated. This site is important for the inactivation if trypsin is aberrantly activated within the pancreas. The mutation makes it impossible to inactivate the enzyme, which predisposes the carrier to pancreatitis.

Any mutation in the gene for the pancreatic secretory trypsin inhibitor, PSTI, can be a hereditary cause of chronic pancreatitis.\(^{22}\) PSTI inhibits trypsin that’s activated within the pancreas as a defense mechanism against pancreatitis. When this defense mechanism doesn’t function, even a small amount of activated trypsin within the pancreas can lead to pancreatitis, since trypsin activation is self-perpetuating. Some mutations in the cystic fibrosis transmembrane conductance regulator (CFTR) gene have been associated with chronic pancreatitis, even in the absence of a diagnosis of cystic fibrosis. The CFTR gene codes for a chloride channel present in pancreatic and lung cells. Interference with normal chloride secretion due to mutations in the CFTR gene causes thick, viscous secretions, which may cause pancreatitis due to blockage. People with cystic fibrosis develop pancreatic insufficiency.
Autoimmune pancreatitis (AIP) is a rare and recently identified, but increasingly recognized, cause of early onset chronic pancreatitis, although the pathology is unclear. There are two types of AIP, which are difficult to distinguish diagnostically from pancreatic carcinoma. What distinguishes AIP from pancreatic carcinoma are increased serum immunoglobulin G (IgG) and/or IgG4; the presence of autoantibodies; imaging that reveals fibrosis; and enlargement and/or ductal narrowing. Otherwise, clinical presentation is similar. Serum testing and imaging techniques, therefore, are crucial to avoid unnecessary pancreatic resection due to an incorrect diagnosis of pancreatic cancer, since AIP can be treated with steroids. AIP is the only known type of pancreatitis that has a successful nonsurgical treatment protocol. The underlying disease processes of AIP and diagnosis and treatment guidelines are being investigated.

Pancreas divisum (PD) is a congenital anomaly that may cause chronic pancreatitis due to obstruction of the normal flow of secretion. Obstructive pancreatitis occurs when pancreatic secretions can’t be emptied into the duodenum. In this environment, pancreatic secretions build up in the pancreatic duct, and trypsin is activated from the pressure that builds up, resulting in tissue damage. PD is relatively common, estimated to be present in 5% to 11% of the US population. During normal embryonic development, two pancreatic ducts fuse to form the main pancreatic duct. In PD, the absence of this fusion leads to two drainage ducts from the pancreas, both of which are narrower than the normal single, fused duct. The narrow ducts, especially when present in conjunction with another risk factor such as a genetic predisposition, increase the likelihood of blocked pancreatic secretions and subsequent pancreatitis. Many people with PD show no clinical manifestations, but a subset of those with PD will develop chronic pancreatitis, which may be treated surgically by pancreatic resection and drainage.

While several causes of pancreatitis have been identified, the etiology of 10% to 30% of cases is unclear and categorized as idiopathic. There are likely genetic mutations and environmental exposures that have yet to be identified that predispose individuals to pancreatitis. In fact, one study conducted genetic sequencing in patients with idiopathic adult onset pancreatitis to search for new genetic mutations. Of the 67 subjects, 44 were found to have a mutation, but most had no family history.

Much is known about the etiology of pancreatitis, but clearly more studies will help to further delineate the exact underlying disease processes for the multiple causes of chronic pancreatitis. Ultimately, this will lead to a higher likelihood of successful treatment and management.

**Symptoms and Diagnosis**

Both acute and chronic pancreatitis cause severe abdominal pain, which is the primary reason patients seek medical care. Acute pancreatitis is easily diagnosed based on elevated serum amylase and lipase, the most sensitive and specific measurements. Chronic pancreatitis is more difficult to diagnose, especially if the disease is in its early stages during which morphologic changes to the tissue may be undetectable, if sufficient digestive enzymes are being produced, and serum levels of digestive enzymes are normal. There’s no universal gold standard for diagnosis of chronic pancreatitis. A pancreatic biopsy would likely be the most
telling, but it isn’t ideal because the diseased tissue may not be uniform throughout the organ and therefore may be missed, and because the biopsy procedure itself can cause acute pancreatitis. Furthermore, there’s no standard scale of morphologic change for diagnosis of chronic pancreatitis.

Recently, the American Pancreatic Association published guidelines on the diagnosis of chronic pancreatitis. Three levels of diagnosis include definitive, probable, or insufficient evidence, based on a diagnostic algorithm of STEP (survey, tomography, endoscopy, and pancreatic function testing). Morphologic changes that would indicate a definitive or probable diagnosis include calcifications in the pancreatic tissue or in the duct, ductal enlargement, and pseudocysts. Morphologic changes may be identified by medical imaging techniques, including abdominal radiographs, abdominal ultrasound, CT scans, and magnetic resonance imaging (MRI)/magnetic resonance cholangiopancreatography (MRCP), each of which has its strengths and weaknesses. Most imaging techniques, including radiographs and ultrasound, can identify the ductal changes and calcifications that are present in advanced chronic pancreatitis. CT scans are more sensitive for identification of changes within the pancreatic tissue. The most sensitive imaging modality is MRI, since it can catch changes that occur earlier in the disease stage, such as those in the side branches of the pancreatic duct. Secretin stimulation MRCP also may be useful to catch earlier changes in chronic pancreatitis. Endoscopic retrograde cholangiopancreatography can identify changes in the pancreatic duct, but it can cause acute pancreatitis, and thus isn’t recommended for diagnostic purposes. Endoscopic ultrasound was found to be very specific and sensitive to morphologic changes consistent with pancreatitis, but findings varied depending on who conducted the sonography.

Functional changes occur in chronic pancreatitis due to the replacement of normal pancreatic acinar cells with fibrotic tissue. Assessing functional changes for the diagnosis of chronic pancreatitis involves measuring digestive enzyme secretion, either directly or indirectly. Measuring enzymes directly is helpful because this is more likely to identify mild and moderate early stage pancreatic malfunction, while indirect measurements of enzymatic byproducts aren’t as sensitive. Identification of the disease in its earlier stages is advantageous so that interventions to either delay or prevent complications may be implemented immediately. Intravenous injection of the hormone secretin to stimulate acinar cells and measuring subsequent pancreatic output of bicarbonate is the gold standard for directly testing exocrine pancreatic function. Fluid volume of pancreatic secretions are collected from the duodenum, and if bicarbonate concentration is less than 80 mM/L, pancreatic secretory function is compromised. Cholecystokinin injections also may be used to stimulate the pancreas, and subsequent lipase concentration of pancreatic secretions measured. Less than 780,000 IU/L of lipase per liter indicates compromised pancreatic secretory function.

The gold standard for diagnosis of steatorrhea, the presence of fat in the stool, which is an indirect measurement of pancreatic exocrine function, is the 72-hour fecal fat test. Steatorrhea usually isn’t present until chronic pancreatitis is more advanced. Patients ingest 100 g of fat per day for three days, and their stool is collected and evaluated. Greater than 7 g of fecal fat per day indicates fat malabsorption. The drawback of this test is that steatorrhea isn’t specific to pancreatitis and could be due to another condition, such as celiac disease or small intestinal bacterial overgrowth. Another measure of fat malabsorption that’s more
convenient and specific to pancreatic insufficiency (PI) is the pancreatic elastase test. Elastase is a pancreatic enzyme normally found in the stool since it isn’t broken down during intestinal passage. If the pancreas is functioning normally by producing adequate digestive enzymes, a certain amount of fecal elastase will be present in the stool. A fecal elastase level of 100 to 200 µg/g of stool indicates moderate PI, while less than 100 indicates severe PI. The drawback of the fecal elastase test is that it isn’t sensitive enough to identify mild PI.

MNT
Aside from pain control, the medical management goals for chronic pancreatitis are mainly related to nutrition. Pain is almost always treated with analgesics, sometimes without success. Surgical approaches to manage chronic pancreatitis include the Puestow procedure to relieve pancreatic duct hypertension, and pancreatectomy with islet autotransplantation; while the latter is still fairly rare, research shows success. PERT also is associated with reduced pain. The use of antioxidants for pain control has shown promise, but since studies have been inconsistent, and especially because they may increase mortality risk, their use for pain control isn’t recommended.

The three main nutritional goals for patients with chronic pancreatitis are consumption of adequate calories, prevention and/or correction of nutrient deficiencies, and successful PERT. Maintenance of adequate calories can be a challenge due to the combination of decreased appetite, increased calorie needs, and malabsorption. People with chronic pancreatitis have been shown to have a 20% to 50% increase in resting energy expenditure. Calorie recommendations for chronic pancreatitis from the European Society for Parenteral and Enteral Nutrition are 35 kcal/kg/day. Historically, low-fat diets were thought to be appropriate for chronic pancreatitis to reduce stimulation of the pancreas; however, the evidence doesn’t support whether or not low-fat diets are effective, and meeting calorie requirements may be difficult without the extra calories from fat. Recommendations for macronutrient composition are 30% of calories as fat, and 1 to 1.5 g/kg of protein daily. Oral nutrition supplements to boost calorie intake may be helpful. Enteral nutrition should be considered if patients are unable to meet calorie needs by mouth, and parenteral nutrition only if enteral nutrition isn’t an option.

Fat malabsorption and steatorrhea usually aren’t detectable until the disease is in its later stages, once fibrotic tissue has replaced a significant amount of normal pancreatic tissue. Because pancreatic lipase is responsible for approximately 90% of fat digestion, fat malabsorption is a greater problem than carbohydrate and protein malabsorption. There are enzymes from glands other than the pancreas that digest carbohydrates and protein, thus digestion of these macronutrients isn’t as compromised as it is with fat during chronic pancreatitis. The greatest concern of fat malabsorption is the loss of calories and compromised fat-soluble vitamin absorption. Vitamin D deficiency may be present in more than 50% of people with chronic pancreatitis, while vitamins A and E deficiencies have been estimated to be relatively low, 3% and 10%, respectively. Annual assessment of serum vitamin levels is recommended so that intervention may be individualized and unnecessary supplementation will be avoided. There’s no consensus regarding vitamin supplementation for chronic pancreatitis, but some guidelines have suggested following the protocols for patients
with cystic fibrosis (PI, and thus fat malabsorption and fat soluble vitamin deficiency, accompany cystic fibrosis).\textsuperscript{46}

Low serum vitamin D and bone disease are more common in people with chronic pancreatitis compared with otherwise healthy individuals.\textsuperscript{47} Prevalence of bone disease in patients with chronic pancreatitis is estimated to be between 39\% and 74\% due to multiple risk factors, including low serum vitamin D, physical inactivity, cigarette use, and malabsorption.\textsuperscript{48,49} A recent meta-analysis of 10 studies and 513 subjects found that 65\% of people with chronic pancreatitis had osteoporosis or osteopenia.\textsuperscript{50} Annual bone density tests, serum vitamin D testing, weight-bearing exercise, and abstinence from alcohol and smoking will help prevent bone disease in patients with chronic pancreatitis, although formal guidelines for management are lacking.\textsuperscript{35} The key in this situation is early diagnosis of chronic pancreatitis itself so that preventive measures may be implemented before the development of bone disease. Unfortunately, chronic pancreatitis may evade diagnosis until its later stages.

PERT is critical for adequate digestion of the dietary macronutrients by people with chronic pancreatitis. The enzymes are porcine-derived and enteric-coated to survive gastric acidity and come in low- and high-dose variations. Normal pancreatic secretion of lipase is approximately 3,000 to 5,000 units per minute after a meal.\textsuperscript{28} Initial PERT recommendations once steatorrhea is clearly present are 1,000 lipase units/kg of body weight per meal, based on the approximation that 2,000 units are needed per gram of dietary fat.\textsuperscript{28} Individual needs vary, but a general rule of thumb is that 25,000 to 75,000 lipase units are needed per meal, and 25,000 units per snack; one-half taken during the meal, and one-half taken at the end of the meal.\textsuperscript{51} Use of endogenous pancreatic enzymes is considered successful if it reduces steatorrhea, minimizes gastrointestinal distress, and prevents calorie and fat soluble vitamin deficiency. PERT won’t fully prevent fat malabsorption or steatorrhea, but should allow for weight maintenance and increased gastrointestinal comfort.\textsuperscript{8} Fiber intake may interfere with PERT and should be reviewed in the case of ineffective PERT.\textsuperscript{28} If the initial dosing regimen doesn’t result in satisfactory results, the recommendation is to increase the dose by two to three times or try different enzymes.\textsuperscript{28}

Diabetes is a later complication of chronic pancreatitis that occurs due to replacement of insulin-secreting islet cells with nonfunctional fibrotic tissue. Approximately 90\% of people with chronic pancreatitis will develop diabetes.\textsuperscript{11} Diabetes due to chronic pancreatitis also is referred to as pancreatogenic diabetes and is characterized by insulin resistance in combination with PI.\textsuperscript{52} Pancreatogenic diabetes is estimated to make up almost one-tenth of all cases of diabetes in the United States, but unfortunately there’s a lack of treatment guidelines specific to this type of diabetes.\textsuperscript{52}

Risk of pancreatic cancer is a concern for people with chronic pancreatitis. A multicenter historical cohort study of 2,015 subjects with chronic pancreatitis found a 4\% risk of pancreatic cancer at 20 years after diagnosis of chronic pancreatitis.\textsuperscript{53} More recently, a meta-analysis found a relative risk of 13.3\%.\textsuperscript{54} Risk of chronic pancreatitis-associated pancreatic cancer is highest for those with early onset chronic pancreatitis.\textsuperscript{54} Although screening for pancreatic cancer in all chronic pancreatitis patients isn’t considered cost-effective, awareness of the increased risk is important.
Conclusion
The 45-year-old man with the diagnosis of chronic pancreatitis is referred to a dietitian. The dietitian conducts a nutrition assessment on the patient and works with an interdisciplinary medical team to assess pancreatic function. Upon finding low serum vitamin D, signs of steatorrhea, PI, and recent weight loss, the dietitian prescribes an oral nutrition supplement and develops a PERT regimen for the patient. The patient follows up regularly to make sure the PERT is effective and that weight loss is avoided. The dietitian also counsels the patient on abstinence from alcohol.

Chronic pancreatitis is a painful disease with major nutrition and quality of life implications. Nutrition intervention, including counseling on abstinence from alcohol if relevant, alleviates the gastrointestinal discomfort and slows disease progression. Research has demonstrated that dietary counseling is effective for preventing malnutrition in patients with chronic pancreatitis. Understanding the nutritional risks of pancreatitis, including malnutrition and diabetes, allows RDs to better assess, intervene, and follow up with individuals with chronic pancreatitis.

—Megan Baumler, PhD, RD, is an assistant professor at Mount Mary University in Milwaukee.

References


Examination

1. Which of the following is true about the inherent physiological protection against pancreatitis?
   A. The inactive pancreatic digestive enzymes are housed in a cellular granule along with lysosomal hydrolases.
   B. Secretory trypsin inhibitor is present within the pancreas to inactivate trypsin if needed.
   C. The pancreatic digestive enzymes are activated by enterokinase in the acinar cells.
   D. The pancreatic duct is large enough so that it does not get blocked and cause pancreatitis.

2. Which of the following is true about alcohol-induced chronic pancreatitis?
   A. Abstinence may delay disease progression.
   B. Continued alcohol use will not impact disease progression.
   C. Consumption of three alcoholic beverages per day for one year is typically enough to cause pancreatitis.
   D. Most alcoholics develop chronic pancreatitis.

3. Which of the following is true about the etiology of chronic pancreatitis?
   A. Smoking increases risk of chronic pancreatitis only if another risk factor is present.
   B. All genetic risk factors have been identified.
   C. Mutations in the trypsinogen gene are the most common known heritable risk factor.
   D. Any mutation in the pancreatic secretory trypsin inhibitor gene protects against pancreatitis.

4. Why is chronic pancreatitis more difficult to diagnose than acute pancreatitis?
   A. There may be undetectable changes in the early stages of chronic pancreatitis, and there are no standards for diagnosis.
   B. All cases of chronic pancreatitis are easily distinguished with magnetic resonance imaging, CT, or endoscopic ultrasound.
   C. There are no procedures or protocols for the diagnosis of chronic pancreatitis.
   D. Most institutions do not have the equipment needed to test for chronic pancreatitis.

5. Which of the following is the first physiologic event in chronic pancreatitis, regardless of etiology?
   A. A rise in serum amylase and lipase
   B. Blockage of the pancreatic duct
   C. The premature activation of trypsin within the acinar cells
   D. Lack of adequate digestive enzymes and subsequent malnutrition

6. Which of the following is true of pancreatic enzyme replacement therapy (PERT)?
   A. It will completely resolve fat malabsorption and steatorrhea.
   B. It should provide relief of gastrointestinal symptoms, help achieve adequate calorie intake, and reduce risk of bone disease.
   C. It is not helpful because stomach acid inactivates the enzymes before they work.
   D. It’s recommended for pain control.
7. Why do most people with chronic pancreatitis develop diabetes and pancreatic insufficiency?
A. Pancreatic secretions are blocked due to gallstones so insulin and digestive enzymes are unable to reach the small intestine.
B. Insulin receptors in the body become resistant, and digestive enzymes are unable to be activated.
C. Beta and acinar cells that normally secrete insulin and inactive digestive enzymes, respectively, are replaced with nonfunctioning fibrotic tissue.
D. The reason patients with chronic pancreatitis develop insulin and pancreatic insufficiency is not known.

8. Why are individuals with chronic pancreatitis at risk of malnutrition?
A. Their calorie needs generally are lower than those in the healthy population.
B. Fat malabsorption makes it difficult to obtain adequate calories and absorb fat-soluble vitamins.
C. The genetic mutations that cause chronic pancreatitis also cause vitamin A deficiency.
D. Chronic pancreatitis is strongly correlated with proteinuria.

9. What is most important for the nutritional care of an individual with chronic pancreatitis?
A. Carbohydrate counting for pancreatogenic diabetes to achieve adequate blood glucose control to prevent long-term complications.
B. A protein intake of 1.5 g/kg to prevent the loss of lean body mass that typically occurs with chronic pancreatitis.
C. Pain management to achieve maximum quality of life.
D. Adequate calories, successful PERT, and prevention of micronutrient deficiency.

10. Why are individuals with chronic pancreatitis at increased risk of bone disease?
A. They're more likely to have low serum vitamin D, fat malabsorption, physical inactivity, and smoke cigarettes.
B. They're unable to absorb calcium due to changes in the small intestine that affect absorptive surface area.
C. A genetic predisposition to chronic pancreatitis usually is clustered with a genetic predisposition to bone disease.
D. They avoid dairy products to prevent aggravation of the disease, which leads to a reduced calcium intake.