Pancreatitis

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INTRODUCTION

The pancreas is a glandular organ that secretes essential substances that aid in digestion and absorption of nutrients. The pancreas has both exocrine and endocrine functions. Endocrine functions include synthesizing glucagon, insulin, and somatostatins. Exocrine functions include secreting enzymes that aid in the digestion of carbohydrates, lipids, and proteins. Some secretions can be made in the gallbladder and connect to the pancreas via the common bile duct. Secretions made in the pancreas directly connect with the small intestine via the ampulla of Vater. Hormones, secretin and CCK, stimulate the pancreas to synthesize and release secretions (1). When alterations within this system arise, pancreatitis may occur. To better understand and treat pancreatitis, disease description, etiology, pathophysiology, medical diagnosis, medical treatment, and medical nutrition therapy must be understood. Understanding all parts of this disease can help improve treatment by decreasing complications and increasing prognosis.

DISEASE DESCRIPTION

Each year 32 to 44 new cases per 100,000 people occur within the United States. Acute pancreatitis is more prevalent and accounted for 274,000 hospitalizations in 2012. Chronic pancreatitis accounts for approximately three to nine cases per 100,000 people (2). Acute pancreatitis is minor inflammation of the pancreas, which usually resolves after adequate treatment. Acute pancreatitis is localized inflammation and rarely involves other tissue regions and organ system. Chronic pancreatitis is permanent damage that results in chronic inflammation, fibrosis, and loss of tissue function. Chronic pancreatitis leads to loss of endocrine and exocrine function and therefore affects the body systemically.

ETIOLOGY

The two main causes of pancreatitis are biliary obstruction and alcoholism. Most often,

women develop pancreatitis due to a biliary obstruction and men due to alcoholism. Biliary obstructions caused by gallstones account for 45% of acute pancreatitis attacks, while excess alcohol consumption accounts for 35% of attacks. Usually, chronic pancreatitis develops as a result of chronic alcoholism or permanent duct stricture. Other causes of pancreatitis could include but are not limited to medications, hypercalcemia, hypertriglyceridemia, heredity, trauma, infection, ischemia, and idiopathic (3). The most prevalent risk factors associated with pancreatitis are gallstones and alcohol consumption. Other risk factors include hypothermia, infections, tumors, hypertriglyceridemia, and trauma (4).

PATHOPHYSIOLOGY

Acute Pancreatitis

Acute pancreatitis has multiple causes but most often is related to biliary obstruction or excessive alcohol consumption. Both causes up regulate activation of digestive enzymes and lead to inflammation. Inflammation of the pancreas leads to edema, due to increased vascular permeability, and pancreatic enzyme malfunction.

Biliary Obstruction

Acute pancreatitis related to biliary obstructions can block the pancreatic duct, bile duct, or both. Most often, gallstones are the cause of biliary obstructions leading to acute pancreatitis. This blockage leads to increasing pressure and accumulation of digestive zymogens. Zymogens are precursor enzymes, like trypsinogen, that require activation and therefore protect the tissue self-digestion. The build up of pressure and zymogens causes certain protective mechanism to be altered. First, certain enzymes, like SPRINK1, inhibit the activation of zymogens within the pancreas. Second, the pancreas contains a low concentration of calcium in order to inhibit certain reactions. With a biliary obstruction, overwhelming amounts of trypsinogen cancel the effects of

SPRINK1. This enzyme cannot inhibit the change from trypsinogen to trypsin. Also, the pancreatic environment begins to increase calcium levels due to the obstruction. Higher levels of calcium will promote the change from trypsinogen to trypsin (5).

Excess Alcohol Consumption

Excess alcohol consumption interferes with hormones that stimulate digestive enzyme production. Alcohol sensitizes the cells to cholecystokinin (CCK)(6). CCK is a hormone that is responsible for the digestion of fat and protein. Excessive alcohol consumption increases the acinar cells sensitivity to CCK. With increased sensitivity, early zymogen activation occurs. Early zymogen activation causes trypsin to break down pancreatic tissue and results in inflammation (7).

Chronic Pancreatitis

Similar to acute pancreatitis, chronic pancreatitis can result from biliary obstruction and excessive alcohol consumption. Chronic pancreatitis causes permanent damage to the pancreas, which leads to chronic inflammation and loss of function.

Biliary Obstruction

In chronic pancreatitis, biliary obstructions are permanent. Strictures of the main pancreatic duct can develop as a consequence of long-term obstruction or severe acute attacks. Also, trauma to the pancreas may lead to pancreatic duct strictures. Strictures will lead to pancreatic necrosis and pseudocyts. Pseudocyts are a collection of fluid that contains enzymes, blood, and necrotic tissue.

Excess Alcohol Consumption

As discussed earlier, alcohol sensitizes the acinar cells to CCK stimulation, leading to early zymogen activation. Also, alcohol metabolites stimulate pancreatic stellate cells. Stellate

cells are the final stage in the fibrosis pathway (2). Stimulating the production of stellate cells leads to early pancreatic fibrosis and loss of function.

Course of the Disease

Acute pancreatitis requires hospitalization but full recovery is likely. Chronic pancreatitis is permanent damage and patients require longer hospitalization and long-term care. The disease will start with local inflammation caused by tissue damage. Inflammation will lead to increased vascular permeability resulting in edema. In acute pancreatitis, the disease usually does not progress further. In chronic pancreatitis, the disease progresses and tissue necrosis and fibrosis occur. As fibrosis continues, enzyme production declines, which leads to weight loss and malabsorption. Systemic inflammation can occur and patients often experience multiple organ dysfunction syndrome (MODS). Pseudocysts can develop which can lead to pancreatic fistulas, ascites, and pleural effusion (8).

MEDICAL DIAGNOSIS

Pancreatitis has distinctive signs and symptoms associated with the disease. Based on these signs and symptoms, physicians would speculate a patient has pancreatitis. With this speculation, serum amylase and lipase levels would be measured. Other diagnostic tools used to determine etiology and severity could include ultrasound and computed tomography (CT).

Sign and Symptoms

Abdominal pain occurs in 50 to 80% of all cases of pancreatitis. The pain starts in the abdominal region and radiates to the middle back. Patients should notice that pain worsens when eating due to the release of digestive enzymes (8). When patients explain this type of pain, palpation of the region may be done in order to rule out kidney infection. Patients may also experience nausea, vomiting, swollen abdomen, and fever. From these signs and symptoms,

physicians will assume pancreatitis and further testing will be done.

Biochemical Levels

Two specific digestive enzymes are used to diagnose pancreatitis: amylase and lipase. Digestive enzymes are released by destroyed pancreatic cells and will reach the bloodstream causing an elevation in serum levels (2).

Amylase

Amylase is a digestive enzyme released by the pancreas that breaks down carbohydrates. The pancreas accounts for 40-45 percent of serum amylase; the salivary glands account for the rest. Amylase levels rise within six to twelve hours but clear quickly from the blood. Amylase is not specific to the pancreas and is not always sensitive to pancreatitis. Normal levels of serum amylase would be 25 to 125 U/L. Levels above this would suggest pancreatitis (2). Because amylase is not 100 percent specific or sensitive to the pancreas, other biochemical levels should be analyzed.

Lipase

Lipase is a digestive enzyme that breaks down lipids. Lipase levels rise within four to eight hours and remain elevated in the blood for a longer period of time. Also, lipase has a greater specificity for pancreatitis than amylase. Normal levels of lipase would be 0-110 U/L. Elevated levels would indicate the presence of pancreatitis (2).

Other Diagnostic Tools

Other diagnostic tests may be necessary to determine the etiology and severity of the disease.

Ultrasound

An ultrasound gives a visual of the pancreas and surrounding tissue and can help determine the cause of the disease. Ultrasounds are used to search for gallstones, dilation of the bile duct, and ascites (2).

Computed Tomography

Computed tomography (CT) is considered the gold standard for diagnosis of pancreatitis. A CT allows you to see diffuse or segmental enlargement of the pancreas. Areas of inflammation and fluid collection can be viewed. A CT scan helps diagnose the disease severity by showing pancreatic necrosis and fibrotic areas (9).

MEDICAL THERAPY

Acute Pancreatitis

Acute pancreatitis requires hospitalization and medical therapy entails fluid resuscitation, pain relief, food restriction, and treating complications. Close monitoring of respiratory, cardiovascular, and renal function is important as fluids are administered. Patients may experience nausea and vomiting related to medication used to control pain and the lack of food intake (10). Some patients have used Chinese medicinal herbs as an alternative therapy for pain. Studies have shown some herbs show positive effects but no strong evidence exists (11). Most patient quickly recover and about 80 percent of patients with acute pancreatitis will be hospitalized for only one week (9).

Chronic Pancreatitis

Chronic pancreatitis treatment focuses on minimizing pain associated with irreversible damage. Chronic pancreatic pain may be managed through endoscopic, surgical, and/or other techniques. Enzyme replacement therapy has been shown to improve pain by reducing the

amount of CCK produced and therefore decreasing pancreatic stimulation. Additionally, therapy focuses on managing other complications associated with chronic pancreatitis. Pseudocysts may be drain endoscopically or surgically. Biliary obstructions may be fixed surgically or stents may be placed to dilate the obstructed duct. Managing this disease is challenging and treatment effectiveness varies depending on disease severity (3).

Prognosis

Prognosis can be determined by using a clinical scoring system. The Ranson score uses eleven signs during the first 48 hours in order to estimate complications and mortality rate. The higher the Ranson score, the higher the incidence of complications, necrosis, infections, and death. A Ranson score lower than two is associated with a 6.5% mortality rate. A score higher than three is associated with a 62% mortality rate.

MEDICAL NUTRITION THERAPY

Nutrition Assessment

A nutrition assessment is crucial since pancreatitis often causes inadequate oral intake and malabsorption. Necessary tools to use in nutrition assessment would be anthropometrics, biochemical, clinical, and dietary. Patient weight, height, BMI, and usual body weight should be monitored to ensure adequate nutrition is being consumed. Biochemical levels of lipase and amylase should be monitored in order to tract disease course. Glucose levels should be checked often because pancreatic dysfunction could lead to inadequate production of insulin. Clinical signs of pain and fatigue should be noted. Finally, dietary history should be obtained in order to assure adequate oral intake. Some patients may be diagnosed with inadequate oral intake related to abdominal pain, nausea, and vomiting, as evidenced by lack of appetite and weight loss.

Acute Pancreatitis MNT

Patients with acute pancreatitis start with nothing by mouth (NPO) for 48 hours. This allows for pancreatic rest while fluids are being administered. Mild cases of acute pancreatitis will remain NPO for five to seven days. For more sever cases, after 48 hours, enteral nutrition is recommended as a continuous feed by nasogastric, nasoduodenal, or nasojejunal. When choosing formula, small peptide based medium chain triglycerides are recommended. Energy requirements are approximately 25 to 35 kcal/kg/day and protein requirements are 1.2 to 1.5 g/kg/day. Parenteral nutrition is not recommended unless oral or enteral nutrition is not tolerated. As patient improves, enteral nutrition should be discontinued and solid foods may slowly be reintroduced until a healthy normal diet can be tolerated (12).

Chronic Pancreatitis MNT

Patients with chronic pancreatitis should consume a regular diet with supplementations for deficiencies. Supplements may include vitamin B12 injections and bicarbonate replacements. Eating smaller, more frequent meals may reduce symptoms of nausea and vomiting. High-fat foods should be avoided because fat stimulates the pancreas and may increase abdominal pain and lead to steatorrhea. Enzyme replacements should be taken with each meal in order to reduce pancreatic stimulation. All alcohol must be avoided because alcohol stimulates pancreatic secretions (12). Fluid and sodium restrictions may be necessary if comorbidities exist.

CONCLUSION

The pancreas is an important organ because of endocrine and exocrine function. The etiology of pancreatitis is most commonly associated with biliary obstructions, related to gallstones, or excess alcohol intake. Although the entire pathophysiology is unclear, pancreatitis is often related to early zymogen activation and CCK hyper-stimulation. Acute and chronic

pancreatitis can lead to serious complications and therefore proper treatment is important. Medical nutrition therapy is essential for pancreatitis patients because the pancreas supplies digestive enzymes and other important hormones. After treatment, acute pancreatitis often leads to a full recovery but chronic pancreatitis requires constant work to control pain and symptoms.

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