Pancreatitis: Disease Overview with Nutritional Implications

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INTRODUCTION

Pancreatitis is inflammation of the pancreas. It is caused by obstructions, organ damage, and alcohol. There are two kinds of pancreatitis, acute and severe. When managed properly, pancreatitis symptoms can subside.

DISEASE DESCRIPTION

Acute Pancreatitis

Acute pancreatitis is an acute inflammatory process of the pancreas with variable involvement of the pancreas, regional tissues around the pancreas, or remote organ systems (1). The severity of the episode can range from mild to severe. Mild pancreatitis is associated with mild discomfort with minimal pancreatic inflammation. Severe pancreatitis is associated with necrotization of the pancreas that leads to multiple organ dysfunction syndrome (MODS) and death if not properly and promptly treated. Acute pancreatitis is seen in 26-44 per 100,000 people in the United States, though this number varies, depending on source and region of study (2,3). This is a 10% increase from 10 years ago. Hospital admissions for acute pancreatitis were 14.5 to 20.7 per 100,000 inhabitants and the prevalence is 3-5%(3).

Chronic Pancreatitis

Chronic pancreatitis is permanent and irreversible damage of the pancreas, with evidence of chronic inflammation, fibrosis, and destruction of exocrine and endocrine tissue. The major cause is alcohol use (1). Chronic pancreatitis is found in approximately 6 to 7 per 100,000 people. This rate has been slowly rising each year. They attribute this increase to possible increase in alcohol consumption. The prevalence of chronic pancreatitis is estimated at 26.4 per 100,000 inhabitants (4).
ETIOLOGY AND RISK FACTORS

Acute Pancreatitis

The two most common causes of acute pancreatitis are gallstones and alcohol abuse; together they represent more than 80% of cases. The risk of developing gallstone related pancreatitis in patients with asymptomatic gallstones is very low, .05%-.2%. The same goes for alcohol consumers. Only a minority of alcohol abusers develop pancreatitis, about 2-3%. Other causes can be structural abnormalities, neoplasms, metabolic disorders, drugs, trauma, iatrogenic causes (i.e. post endoscopic retrograde cholangiopancreatography [ERCP] pancreatitis), infections, vascular disorders, genetic causes (i.e. trypsinogen mutations), and the rest, that don’t fall into any of these categories, are classified as idiopathic induced pancreatitis (1,3).

Chronic Pancreatitis

The causes of chronic pancreatitis are divided into three categories: alcohol, idiopathic, and ‘other’. Alcohol is by far the leading cause accounting for approximately 70-80% of all cases. 20% of cases are related to idiopathic causes and the remaining 10% fall under the ‘other’ category. The other category encompasses cases associated with hyperparathyroidism, hypertrygliceridemia, duct obstruction, trauma, pancreas divisum, autoimmune pancreatitis and hereditary pancreatitis (4).

PATHOPHYSIOLOGY AND COURSE OF THE DISEASE

Acute Pancreatitis

Acute pancreatitis is the result of a series of pathological events. Initially it involves the acinar cell, reduced blood flow, and/or impairment of enzyme secretion. There are two levels of acute pancreatitis, mild and severe (5). They are determined by the severity of destruction and damage the episode of pancreatitis does to the pancreas and surrounding organs. Once the
inflammatory process of acute pancreatitis is initiated it can involve the whole pancreas, surrounding tissues, and causes a systemic reaction that harms many organs.

Acinar cells are the functional unit of the exocrine pancreas. They synthesize, store and secrete digestive enzymes (6). If the acinar cells do not function properly, or if the pancreatic enzymes that are supposed to be secreted are activated prematurely, the acinar cells in the pancreas are damaged or destroyed. This damage leads to necrosis, hemorrhage and inflammation of the gland. Increased capillary permeability can lead to edema and decreases the blood supply to the pancreas which further complicates the episode (1,6).

Endothelial injury, vasospasm, and vascular thrombosis can all occur during the episode of acute pancreatitis and can change pancreatic perfusion. This leads to reperfusion injury and free radical generation. Ischemia and loss of perfusion can directly lead to cell death. Acinar cells and duct cells (cells that line the pancreas) may also lose their tight junctions because their ‘anchor’ cells, actin cytoskeleton, breakdown. This breakdown allows pancreatic duct contents to leak into the interstitial space. The leakage of fluids leads to a rapid increase in serum levels of pancreatic enzymes and a decrease in pancreatic secretion.

These rapid changes in the pancreas lead to the initiation of the inflammatory response. Neutrophil recruitment and activation, TNF, and platelet-activating factor are generated by the acinar cell and stimulate inflammation. This inflammatory process can lead to MODS or severe inflammatory response syndrome, sepsis, and if not treated appropriately, death (4).

**Chronic Pancreatitis**

Chronic pancreatitis is a syndrome of destructive inflammatory conditions (7). It starts with a sentinel (unexpected but injurious) event in the pancreas that triggers an inflammatory process within the pancreas. This ‘event’ can come from a number of causes the biggest two
being alcohol and ducal obstructions (gallstones). Alcohol causes damage by altering cellular membranes and gallstones block the secretions from the acinar cells, causing metabolic alterations in acinar cells and producing oxidative stress molecules. This process is the same one disused in acute pancreatitis pathophysiology. If this process is repeated or prolonged it leads to the activation of pancreatic stellate cells that produce an extracellular matrix (8). This results in a loss of glandular tissues (acinar and duct cells) that are replaced with pancreatic fibrosis (1,7,8). This fibrosis results in decreased number of cells to make a secrete enzymes and also can for a ductal plug leading to further inflammation and destruction.

METHODS OF MEDICAL DIAGNOSIS

Acute Pancreatitis

The first signs of acute pancreatitis are a sudden onset of abdominal pain, nausea, and vomiting. If a patient is exhibiting these initial clinical features, labs and imaging tests are preformed and assessed using the Ranson’s Criteria to determine the presence of pancreatitis and severity. A Ranson’s Criteria assessment is performed upon admission and again after 48 hours.

Serum amylase and lipase are only two of many enzymes made it the pancreas but they are the easiest and quickest to measure. Elevation above three times the normal value is a major indicator that acute pancreatitis is present. Serum lipase often remains elevated longer, which makes it a more useful diagnostic tool after symptoms have subsided (1).

Computed Tomography (CT) scans are also used to diagnose and manage acute pancreatitis; however, not every patient requires one. A CT scan is mainly used to indicate if the initial diagnosis is in doubt or for prognostic purposes in severely ill patients (1,9).

Chronic Pancreatitis

Diagnosis of chronic pancreatitis is very different from acute. Clinical manifestations do
not provide enough diagnostic information to make an accurate diagnosis. Several tests must be performed to diagnose the disease as well as project an accurate prognosis. These tests look at function and structure of the pancreas.

A clinical sign is severe abdominal pain with possible nausea and vomiting. The pain can be intermittent or constant. Other major indications of pancreatitis are: steatorrhea and subsequent weight loss, diabetes mellitus, and clay colored stools.

There are a variety of tests done on patients to determine the presence and severity of chronic pancreatitis (9). These include:

- **CT scans**- to rule out other diseases that have similar clinical manifestations, identify calcification, and identify specific etiologies in more advanced stages.
- **Abdominal Ultrasound**- to measure pancreatic duct dilation and identify morphological (size and shape) changes in the pancreas. The images are used in diagnosis to establish the severity of the disease and determining the most appropriate treatment.
- **Magnetic Resonance Imaging (MRI)**- examines parenchyma before and after intravenous gadolium administration, looks at the duct system, evaluates pancreatic exocrine function. Sensitive means for diagnosing even in the early phases of the disease.
- **ERCP**- invasive imaging procedure that cytology and biopsy tissue sampling, visual inspection of ducts. Operator-dependant procedure that requires training, experience and associated with low failure and complication rates. Used often in therapeutic options such as relief of pancreatic ductal obstruction from stones and/or strictures.

**CURRENT MEDICAL THERAPIES**

**Acute Mild Pancreatitis**

Treatment of mild acute pancreatitis is mainly supportive care to relieve symptoms. This
includes aggressive administration of IV fluids, pain medications, antiemetics, and bowel rest until nausea and vomiting have stopped (10). Patients are usually given nothing by mouth (NPO) for 3-5 days. In this short period of time, many doctors do not initiate tube feedings or parenteral feedings because of the increased risk for infection. Studies also show that insufficient nutrition in this brief time does not negatively influence outcomes (1). Other medical treatments may include:

- **Nasogastric suction**: used when vomiting. Prevents hormone-stimulated pancreatic secretion by removing acid from stomach before it reaches duodenum. Only used if needed for other problems (bowel obstruction, ileus or intractable vomiting).
- **Acid suppression**: proton pump inhibitors to decrease acid-induced pancreatic secretions.
- **Somatostatin/Octreotide**: hormone that suppresses pancreatic exocrine function directly by depressing pancreatic exocrine secretion and indirectly by suppressing gastrointestinal hormone secretion.
- **Gabexate mesilate**: Protease inhibitor (enzyme released from pancreas that breaks down proteins).

Treatments are administered until symptoms subside.

**Acute Severe Pancreatitis**

Treatments for severe pancreatitis are the same as mild with the addition of nutrition support. Patients are given enteral feedings after 3-5 days and may be given parenteral support depending on the doctor and hospital’s protocol for PN and TPN use. Some studies suggest that antioxidants may be an appropriate form of treatment because of damage caused by oxidative stress during the inflammation episode but there is not strong enough evidence to support it (11).
Chronic Pancreatitis

Because alcohol is a major cause of chronic pancreatitis, the most important treatment of chronic pancreatitis is cessation of alcohol use. Other treatments used to decrease symptoms are: low-fat diet and small meals, analgesics to manage pain, pancreatic enzyme preparations to decrease pancreatic enzyme production, decompression or the drainage of fluid buildup around the pancreas, H₂ receptor antagonists or proton pump inhibitor to decrease acid, somatostatins to decrease enzyme production, cholecystokinin (CCK) antagonists to suppress amylase and trypsin secretions (1), endoscopic and surgical treatments, and vitamin supplementation (A,D,E,K, and B12).

APPROPRIATE TOOLS TO USE IN NUTRITION ASSESSMENT

Patients with acute and chronic pancreatitis are NPO for the first 3-5 days upon admission to the hospital for bowel rest (12). When a patient stays beyond 5 days the initiation of nutrition support becomes an item of concern. The patient’s medical team decided when to initiate nutrition support based to the patient’s lab values, disease prognosis, and overall health status. Specific lab values of concern are decreased concentrations of free amino acids and intracellular glutamine and a marked elevation of aromatic amino acids (12). Patients with pancreatitis, especially chronic pancreatitis are at an increased risk for vitamin and fat malabsorption and lab values and clinical signs for deficiencies need to be monitored regularly.

MNT

Acute Pancreatitis

Patients should be NPO and receive fluid support with IV fluids. If oral nutrition cannot be started in 5-7 days, nutrition support should be started. Patients with less severe cases of prolonged acute pancreatitis, tubal feeding can be initiated beyond the ligament of Treitz using a
polymeric formula. Patients with acute pancreatitis are candidates for parenteral nutrition depending on hospital and doctor preferences and policies (12,13). When an oral diet is initiated it should be low-fat (to decrease pancreatic secretions and avoid steatoreia, easily digestible foods, small and frequent meals, meet protein needs, and increase calories to account for malabsorption of nutrients.

Chronic

Patients with chronic pancreatitis have the same guidelines for NPO upon admission and oral nutrition requirements are the same. Patients that cannot meet their nutrition needs orally are given either enteral or perenteral nutrition support. Patients often need pancreatic enzymes to decrease secretions and need vitamin supplements for fat-soluble vitamins and vitamin B12 along with bicarbonate (12).

LONG TERM PROGNOSIS

Acute Pancreatitis

Most cases of acute pancreatitis go away in about a week. If the not treated properly it can develop into a life-threatening illness. Pancreatitis can return, the likelihood depends on the cause and the success of the treatment. If a patient experiences severe acute pancreatitis they are at a higher risk of experiencing hemorrhagic pancreatitis, liver, heart or kidney impairment, and/or necrotizing pancreatitis. These complications increase the mortality (4,10,12).

Chronic

Because chronic pancreatitis is a progressive and irreversible loss of pancreatic structure and function they may never have full function return to their pancreas. Many patients undergo surgery which increases risk for infection and mortality rates increase. When initial medical and endoscopic treatments fail to relieve abdominal pain further surgery may be required.
Pancreatitis can result in disability or death if not treated quickly and appropriately (10,12).
REFERENCES

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