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Acute Pancreatitis

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Introduction

Acute pancreatitis is a common diagnosis seen in intensive care units worldwide. The incidence of pancreatitis has increased over the last decade. It ranks third amongst the gastrointestinal diseases resulting in hospital admissions. The destructive complications of pancreatitis make it a life-threatening disease. If pancreatitis progresses to the severe form the mortality rate significantly increase from one percent to upwards of thirty percent. Pancreatitis is associated with high rates of morbidity, mortality, and prolonged hospital admissions(Goozen, Besselink, Santroort, & Bollen, 2013). An increased understanding of the pathophysiology of pancreatitis has changed the approach to treatment from early surgical treatment to a more conservation and all encompassing approach utilizing antibiotic therapy, multidisciplinary team involvement, early nutrition, and other forms of supportive care. A majority of the supportive care is provided directly by the bedside nurse(Sahora, Jakesz, & Gotzinger, 2009). This topic was chosen to increase nurses' knowledge of the pathophysiology of pancreatitis, the presentation of the disease symptoms, the treatment, and the implications that the care provided can have on patient outcomes.

Epidemiology

In the Western World Acute Pancreatitis (AP) affects approximately 40/100,000 people yearly. Eighty percent of those suffering from AP will experience a self-limiting disease process, while the other twenty percent develop a severe and highly deadly form of AP. The two most common causes of adulthood AP are gallstone and alcohol-induced AP (Harper & Cheslyn-Curtis, 2011). AP affects men and women equally, however men are more likely to develop chronic pancreatitis. AP is more common amongst African Americans than Caucasian Americans. The severity of AP increases with age, and those less than twenty years of age are less likely to develop AP. Risk factors such as obesity, alcoholism, smoking, high fat diets, and genetic predisposition increase the incidence and severity of pancreatitis. (Yadav & Lowenfels, 2013).

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Pathophysiological Processes

Signs and Symptoms

The signs and symptoms of AP vary according to the severity of the patient's illness. There is the mild from of AP, also known as interstitial pancreatitis, moderate pancreatitis, and the severe pancreatitis. The hallmark-presenting symptom in all forms of AP is abdominal pain. The location is typically epigastric, right upper quadrant, or pain that radiates to the back. The pain may be constant, intermittent, or amplified after eating. Additional signs and symptoms are summarized in the table below (Penny, 2012).

Signs	Symptoms
Elevated lipase, amylase, trypsin, WBC	Abdominal pain
Abdominal distension	Back pain
Absent bowel sounds	Nausea
Fever	Vomiting
Tachycardia	Diarrhea
Tachypnea	Anorexia
Hypotension	
Jaundice	
Cullen's sign	
Grey-Turner's sign	

Causes

- Gallstones
- Alcohol consumption
- Pancreatic obstruction
- Trauma
- Infections
- Genetic mutations of PRSSI, SPINK, CFTR
- Hypertriglyceridemia
- Hypercalcemia
- Autoimmune
- Iatrogenic (ERCP, cardiopulmonary bypass)
- Idiopathic
- Drugs (HCTZ, steroids, azathioprine, furosemide, Bactrim)

(Harper & Cheslyn-Curtis, 2011).

Pathophysiology

The pancreas functions as both an endocrine and exocrine gland. Ninety-eight percent of the pancreas is composed of exocrine cells known as acinar cells. Two percent function as endocrine cells and are known collectively as the islet of Langerhans. The islet cells produce insulin, glucagon, and somatostatin The Acinar cells produce the digestive proenzyme trypsinogen and 15 other digestive enzymes. The digestive enzymes remain inactive in zymogens until they are released into the duodenum to aid in digestion of fats, proteins, and carbohydrates. These digestive juices enter the duodenum via the ampulla of vater. In the duodenum trypsinogen is converted to trypsin and triggers a cascade activating the remaining digestive enzymes (Bakoyiannis, Delis, & Dervenis, 2010). Low intracellular PH, low intracellular calcium , pancreatic trypsin inhibitor, and protease-activated receptor 2 all function as innate protective mechanisms inside the zymogens to prevent early activation of digestive enzymes (Harper & Cheslyn-Curtis, 2011).

Pancreatitis is inflammation of the pancreas that is caused by a disruption in cellular homeostasis from an over stimulation of pancreatic activity and a failure of protective mechanisms. While the exact underlying pathophysiology of pancreatitis is unclear the initiating injury results in either damage to the acinar cells or impaired secretion of digestive enzymes (Hasibeder, Torgersen, Reiger, & Dünser, 2009). Once pancreatic injury has occurred the following maladaptive processes take place:

- Fusion of lysosomal and zymogen compartments
- Activation of trypsinogen to trypsin
- Trypsin activation of remaining zymogen proenzymes
- Activated enzymes digest pancreatic tissue
- Edema, vascular permeability, and hemorrhage ensue
- Pancreatic cell death by necrosis and apoptosis

Inflammation and cell death trigger a massive inflammatory response. Neutrophils and macrophages migrate to site of injury via signaling from chemokines. Cytokines such as: interleukin-6, interleukin-10, and tumor necrosis factor, and reactive oxygen species are produces by macrophages and neutrophils further perpetuating the inflammatory response and leading to a systemic inflammatory process. Increased vascular permeability allow translocation of inflammatory mediators and bacteria into systemic circulation putting patients at increased risk of developing sepsis (Bhatia et al., 2005).

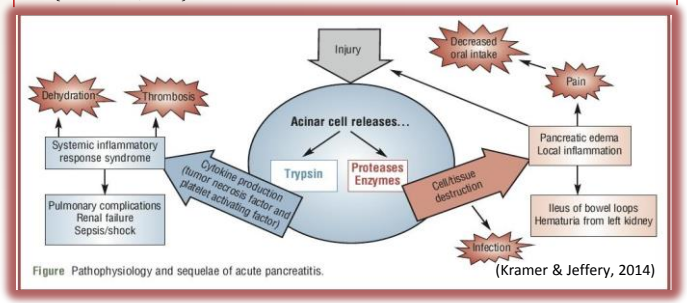

Figure Pathophysiology and sequelae of acute pancreatitis. (Kramer & Jeffery, 2014)

Significance of Pathophysiology

Those patients suffering from acute pancreatitis are at risk of developing life threatening complications. These complications are the direct result of the systemic inflammatory response inducted by pancreatitis. The extent of the systemic inflammatory response directly correlates with the severity of pancreatitis. Understanding the pathophysiology is the key to providing goal directed therapy. The underlying cause must be identified, and corrected if possible, to stop the inflammatory response.

Implications for Nursing

Early recognition and diagnosis of pancreatitis is key to preventing disease progression and associated complications. Advanced practice nurses (APN) must be able to complete a through history and physical in order to correctly identify pancreatitis, and to eliminate differential diagnoses of similar presentation. The APN must be able to identify risk factors associated with pancreatitis, have astute assessment skills, and know what diagnostics tests to order. In order to prevent disease progression the cause of pancreatitis must also be identified (Andris, 2010).

Once a diagnosis of pancreatitis has been made the APN must assess the severity of the disease process. It is vital to correctly assess the severity of the disease process to ensure proper patient placement in the hospital. Sever forms of pancreatitis must be recognized and patients transferred to an intensive care unit as soon as possible. The severity is determined using a combination of assessment data, test results, and a severity index tool (Andris, 2010).

The Ranson's criteria assessment scale assess a patient on admission and again at 48 hours post admission. It can reliably indicates severity, risk or organ failure, and mortality. The presences of 3 or more Ranson's signs is indicative of severe pancreatitis. The more Ranson's signs a patient has strongly correlates with mortality. Listed below is the Ranson's criteria (Sargent, 2005)

On presentation
- Age >55
- WBC > 16,000/mm3
- Blood Glucose > 11 mmol/liter
- Lactate dehydrogenase> 400IU>liter
- AST>25O IU/liter

48 hours post onset
- HCT decreased by 10%
- Blood urea >1.8mmol/liter
- Calcium < 2 mmol/liter
- PaO2 < 80%
- Base deficit >4 mmol/liter
- Fluid deficit > 6 mmol/liter

Risk Factors
- 0-2 <1% mortality
- 3-4 15% mortality
- 5-6 40% mortality
- >6 100% mortality

Those patients with a score of less than 3 have a mild and usually self-limiting disease course. Medical management is aimed at NPO status for pancreas and bowel rest, fluid resuscitation, pain management, electrolyte replacement, anti-emetic administration, glucose control, nutritional support, antibiotic coverage is indicated, and monitoring for development of complications (Sargent, 2005).

Those patients with a score of 3 or greater are at a high risk of developing serious complications and require aggressive treatment in addition to the supportive care given in mild pancreatitis. These patients suffer from severe third spacing resulting in hypovolemia. Hypovolemia not corrected by fluid resuscitation causes decreased perfusion and leads to multi-organ failure. Multiple system organ failure often causes mortality. These patients often require mechanical ventilation for the treatment of acute respiratory distress syndrome, vasopressor support for shock, and dialysis for acute renal failure. These patients are often septic which further complicates the treatment course. Severe pancreatitis can progress to necrotizing pancreatitis (NP). Sterile NP it treated conservatively with antibiotic therapy until the systemic illnesses can be resolved. Early surgical treatment of sterile NP increases risks of complications. Infectious NP is treated with surgical debridement of the pancreases (Andris, 2010).

Nursing care must be directed at preventing complications, and optimizing patient outcomes. This can be done by careful monitoring of the patient's condition. Nursing care must also providing preventive interventions to avoid catheter-related blood stream infections, catheter-associated urinary tract infections, ventilator-associated pneumonia, and pressure ulcers to combative preventable complications (Hofmeyr, Warren, & Van Rensburg, 2014).

Conclusion

Pancreatitis is a common diagnosis seen in intensive care units. It can be mild and self-limiting, or progress to a life threatening disease state. Gallstone and alcoholic pancreatitis are the most common causes of pancreatitis. Patients may present with varying degrees of symptoms. Without the astute care of and constant monitoring by nursing staff, patients are at risk of developing severe complications resulting in death.

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