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Acute Pancreatitis
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Introduction
Acute pancreatitis is a common disease seen in intensive care units worldwide. The incidence of pancreatitis is increasing, and the mortality rate is not decreasing. It ranks third among the gastrointestinal diseases resulting in hospital admissions. The destructive complications of pancreatitis can lead to a life-threatening disease. If pancreatitis progresses to the severe form the mortality rate is significantly elevated from one percent to upwards of thirty percent. Pancreatitis is associated with increased rates of morbidity, mortality, and prolonged hospital admission(Gossens, Sahora, Jakesz, & Gotzinger, 2010; Sahora, Jakesz, & Gotzinger, 2009). An increased understanding of the pathophysiology of pancreatitis has changed the approach to treatment from early surgical correction to a more aggressive and all encompassing approach utilizing antibiotic therapy, enteral/parenteral nutrition, early and continuous monitoring and all forms of supportive therapy. A majority of the supportive care is provided directly by the bedside nurses(Sahora, Jakesz, & Gotzinger, 2009). This topic was chosen to increase nurses’ knowledge of the pathophysiology of pancreatitis, the presentation of the disease, the symptoms, the treatment, and the complications that the care provider can have on patient outcomes.

Pathophysiology

Pathophysiology

The pancreas functions as both an endocrine and exocrine gland. Ninety-eight percent of the pancreas is composed of exocrine cells known as exocrine cells. Two percent of the pancreas function as endocrine cells and are known collectively as the islets of Langerhans. The insulin cells produce insulin, glucagon, and somatostatin. The Acinar cells produce the digestive protease trypsinogen and 15 other digestive enzymes. The digestive enzymes remain inactive in zymogens until they are secreted into the duodenum to aid in digestion of fats, proteins, and carbohydrates. These digestive enzymes enter the duodenum via the ampulla of Vater. In the duodenal trypsinogen is converted to trypsin and triggers a cascade activating the remaining digestive enzymes (Harper & Chenlyn-Curtis, 2011). Inflammation and cell death trigger a massive inflammatory response. Neutrophils have occurred the following maladaptive processes take place: 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 events in injury to damage the acinar cells or impaired secretion of digestive enzymes (Haasbeller, Torgersen, Rieger, & Dünser, 2005). Once pancreatic injury has occurred & metabolic response directly correlates with the severity of pancreatitis. Understanding the inflammatory response inducted by pancreatitis. The extent of the systemic inflammatory response and leading to a systemic inflammatory process. Increased inflammation and cell death trigger a massive inflammatory response. Neutrophils and macrophages produce a site of injury via signaling from cytokines. Cytokines such as interleukin-6, interleukin-10, and tumor necrosis factor, and reactive oxygen species are produced by macrophages and neutrophils further perpetuating the inflammatory response. In a systematic inflammatory process, increased vascular permeability allows translocation of inflammatory mediators and bacteria into systemic circulation putting patients at increased risk of developing sepsis (Bhutta et al., 2005).

Causes

- Gallstones
- Alcohol consumption
- Pancreatic obstruction
- Trauma
- Infections
- Genetic mutations of PRSS1, CFTR, CYFTR
- Hypercalcemia
- Hypertension
- Autoimmune
- Lactogen (Igf2, FSH, thyroid, sex hormone)
- Idiopaths

References


Bhutta, E. S. (2005). Acute Pancreatitis

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 thorough history and physical in order to identify patients at risk, and eliminate differential diagnoses of similar presentation. The APN must be able to identify risk factors associated with pancreatitis, and use a series of assessment tools to test for the diagnosis. In order to prevent disease progression the cause of pancreatitis must be identified and treated. Once a diagnosis of pancreatitis has been made the APN must assess the severity of the disease. It is vital to correctly assess the severity of the disease process in order to determine the appropriate level of care. Fewer forms of pancreatitis are recognized and patients transferred to an intensive care unit as soon as possible. The severity of the condition directly correlates with the severity index tool (Andris, 2010).

The goal of nursing care is to provide a safe environment for the patient and prevent early activation of digestive enzymes (Harper & Chenlyn-Curtis, 2011). The hallmark of the presentation of the pancreas 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 events in injury to damage the acinar cells or impaired secretion of digestive enzymes (Haasbeller, Torgersen, Rieger, & Dünser, 2005). Once pancreatic injury has occurred & metabolic response directly correlates with the severity of pancreatitis. Understanding the inflammatory response inducted by pancreatitis. The extent of the systemic inflammatory response and leading to a systemic inflammatory process. Increased inflammation and cell death trigger a massive inflammatory response. Neutrophils and macrophages produce a site of injury via signaling from cytokines. Cytokines such as interleukin-6, interleukin-10, and tumor necrosis factor, and reactive oxygen species are produced by macrophages and neutrophils further perpetuating the inflammatory response. In a systematic inflammatory process, increased vascular permeability allows translocation of inflammatory mediators and bacteria into systemic circulation putting patients at increased risk of developing sepsis (Bhutta et al., 2005).

Significance of Pathophysiology

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

References


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, patient outcomes can suffer. These patients suffering from acute pancreatitis are at risk of developing life threatening complications. These complications are the direct result of the systemic inflammatory response induced by pancreatitis. The extent of the systemic inflammatory response directly correlates with the severity of pancreatitis. Understanding the pathophysiology is the key to preventing and directing therapy. The underlying cause of pancreatitis must be identified, and corrected if possible, to stop the inflammatory response.