Acute Pancreatitis

Laura Payne  
*Otterbein University, laura.payne1@otterbein.edu*

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Acute Pancreatitis
Otterbein University, Westerville, Ohio
Laura Payne RN, BSN

Introduction

Acute pancreatitis is a common disease 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 can lead to a life threatening disease. If pancreatitis progresses to the severe form hospital mortality rates significantly increase from one percent to upwards of thirty percent. Pancreatitis is associated with a high rate of morbidity and mortality, and prolonged hospital admission(Gossens, Sahora, Bollen, Sargent, & Gooszen, 2005). An increased understanding of the underlying pathophysiology of pancreatitis has changed the approach to treatment from early surgical treatment to a more conservative and all encompassing approach utilizing antibiotic therapy, nutritional and supportive treatment, 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).

Pathophysiological Processes

Signs and Symptoms

The signs and symptoms of AP vary according to the severity of the patient’s illness. There are three different types of AP based on clinical presentation, moderate pancreatitis, and severe pancreatitis. The hallmark symptoms of pancreatitis is 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).

<table>
<thead>
<tr>
<th>Signs</th>
<th>Symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>Elevated lipase, amylase, trypsin, WBC</td>
<td>Abdominal pain</td>
</tr>
<tr>
<td>Fever</td>
<td>Vomiting</td>
</tr>
<tr>
<td>Tachycardia</td>
<td>Anorexia</td>
</tr>
<tr>
<td>Hypotension</td>
<td>Jaundice</td>
</tr>
<tr>
<td>Cullen’s sign</td>
<td>Grey-Turner’s sign</td>
</tr>
</tbody>
</table>

Pathophysiology

The pancreas functions as both an endocrine and exocrine gland. Ninety percent of the pancreas is composed of exocrine cells known as acinar cells. Two percent of the acinar cells produce insulin, glucagon, and somatostatin. The Acinar cells produce the digestive enzymes trypsinogen and 15 other digestive enzymes. The digestive enzymes remain inactive in zymogens until they are activated exocytosed in and digested by free enzymes, proteins, and carbohydrates. These digestive enzymes 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 (Bhatia, Bramot, Dils, & Denvir, 2010). Low intraluminal pH, low intracellular calcium, pancreatic trypsin inhibitor, and protease- activated repressor 2 all function as innate protective mechanisms to prevent early activation of digestive enzymes (Harper & Chenkyn-Curtis, 2011).

Inflammation is the activation of the pancreas that is caused by a disruption in cellular homoeostasis 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 (Faulkner, Torgersen, Reiger, & Dünser, 2009). Pancreatic injury has occurred & may show a collection of blood and necrosis (Hofmeyr, Warren, & Van Der Merwe, 2010). The APN must be able to identify risk factors and correlate them with patient’s signs to prioritize level of care (Hoblitzel, Torgesen, Rieger, & Dünser, 2009). Risk factors of developing pancreatitis are shown in the table below (Sargent, S., 2014).

Risk Factors

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

Patients with a score of less than 3 will have a mild and usually self-limiting disease. Those patients with a score between 3 and 4 will have moderate pancreatitis. Those patients with a score of 5 or more will have severe pancreatitis, and are at a high risk of developing serious complications. The APN must be able to complete Ranson’s criteria to identify patient’s at risk of developing serious complications (Hoblitzel, Torgersen, Rieger, & Dünser, 2009).

Inflammation and cell death trigger a massive inflammatory response. Neutrophils and macrophages migrate to 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. As a result of the inflammatory process, increased vascular permeability allows translocation of inflammatory mediators and bacteria into systemic circulation putting patients at increased risk of developing sepsis (Bhatia et al., 2005).

Causes

- Gallstones
- Alcohol consumption
- Sudden hypotension
- Trauma
- Infections
- Genetic mutations of PRSSI, SPINK, CFTR
- Hypothyroidism
- Hypertension
- Autoimmune
- Lactogen (IREP, cardiolipin antibodies)
- Idiopaths

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 induced by pancreatitis. The extent of the systemic inflammatory response directly correlates with the severity of pancreatitis. Understanding the pathophysiology is the key to providing directed therapy. The underlying cause of pancreatitis 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 thorough history and physical in order to strictly identify pancreatitis, and eliminate differential diagnoses of similar presentation. The APN must be able to identify risk factors and correlate them with patient’s signs to prioritize level of care (Sargent, S., 2014). This information aids the APN in determining if a patient requires admission to the intensive care unit or if more Ranson’s signs is an indication of serious pancreatitis. The more Ranson’s sign a patient’s signs strongly correlates with mortality. Listed below is the ranson’s criteria (Sargent, S., 2006).

48 hours post onset
1. WBC > 100,000/ml
2. Blood urea > 12mmol/l
3. Lactate dehydrogenase > 400IU/l
4. AST > 250IU/l
Risk Factors

48 hours post onset
1. Blood urea >18mmol/l
2. Blood lactic acid 2mmol/l
3. PaO2 < 80mmHg
4. PaCO2 > 6mmHg

Infections related blood stream infections, cancer, and radiation associated blood stream infections, ventilator-associated pneumonia, and pressure ulcers to combative preventable complications (Hofmeyr, Warren, & Van Der Merwe, 2010). The APN must be able to complete Ranson’s criteria to identify patient’s at risk of developing serious complications. The APN must be able to complete Ranson’s criteria to identify patient’s at risk of developing serious complications (Hoblitzel, Torgersen, Rieger, & Dünser, 2009).

References

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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 cause 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 including death. (Bhatia, M., Wong, L., Tan, Y., Huang, J., Panel, P., & Delves, C.,(2005). Acute pancreatitis. Pathophysiology of acute pancreatitis. Langenbeck’s Archives Of Surgery/ Deutsche Gesellschaft Für Chirurgie, 350, 527-539.)